SKIN CIRCULATION IN AREAS PRONE TO PRESSURE ULCERATION AND ADJACENT NONSUSCEPTIBLE AREAS AS DETERMINED BY THE THERMAL RECOVERY METHOD

by

Vern Hedegaard

A thesis submitted to the faculty of
The University of Utah
in partial fulfillment of the requirements for the degree of

Master of Science

College of Nursing

The University of Utah

March 1984

THE UNIVERSITY OF UTAH GRADUATE SCHOOL

SUPERVISORY COMMITTEE APPROVAL

of a thesis submitted by

Vern Hedegaard

This thesis has been read by each member of the following supervisory committee and by majority vote has been found to be satisfactory.

Chairman: Sue E. Huether, R.N., Ph.D.

Mary Ann Johnson, R.N., M.S.

James S. Wood, M.D.

THE UNIVERSITY OF UTAH GRADUATE SCHOOL

FINAL READING APPROVAL

To the Graduate Council of The University of Utah:

I have read the thesis of Vern Hedegaard in its final form and have found that (1) its format, citations, and bibliographic style are consistent and acceptable; (2) its illustrative materials including figures, tables, and charts are in place; and (3) the final manuscript is satisfactory to the Supervisory Committee and is ready for submission to the Graduate School.

Sue E. Huether, R.N., Ph.D.
Member, Supervisory Committee

Approved for the Major Department

Linda K. Amos, F.A.A.N.
Chairman, Dean

Approved for the Graduate Council

ABSTRACT

Pressure ulcers, also called decubitus ulcers and bed sores, are areas of hypoxemic necrosis resulting from compression of capillaries by externally applied pressure. They most commonly develop in areas of the body subjected to prolonged high pressure, such as the trochanters, sacrum and ischia. Very little is known about the local skin circulation in these areas. Reduction of the magnitude and duration of pressure applied to the body surface have been shown to be effective, scientifically based nursing interventions. Interventions based on increasing skin blood flow, although commonly used, have not been shown to be effective or scientifically based.

The purpose of this study was to compare the skin circulation of areas prone to pressure ulceration with the skin circulation of adjacent, less susceptible areas. The thermal recovery method was used to estimate the skin blood flow over the trochanter and sacrum, and compared to that over the abdomen and groin.

No significant differences were found between the thermal recovery values of the trochanter and groin,

or between those of the sacrum and abdomen. Although subjects' thermal recovery values varied widely, no significant differences were observed based on site, age, sex, diastolic blood pressure, or height/weight ratio.

The results of this study indicate a need for continued study of the skin circulation of areas prone to pressure ulceration. The conclusions of other studies utilizing skin temperature as an index of circulation require reexamination. The emphasis in nursing intervention should continue to be directed toward relieving pressure, rather than increasing skin circulation.

CONTENTS

ABSTRACT. iv

LIST OF TABLES. viii

ACKNOWLEDGMENTS x

Chapter

I. INTRODUCTION AND LITERATURE REVIEW 1

Introduction 1
Review of the Literature 5

II. CONCEPTUAL FRAMEWORK 29

Assumptions. 29
Conceptual Framework 29
Research Questions 32
Operational Definitions. 32

III. METHODOLOGY AND RESEARCH DESIGN. 34

Aim. 34
Population 34
Design 35
Instruments. 35
Data Collection Procedures 37
Data Recording 38
Overview of Data Collected 41

IV. DATA ANALYSIS. 43

Methods of Data Analysis 43
Descriptive Statistics 43
Paired t-Test. 44
Results of Data Analysis 46
Discussion 66
Implications for Nursing Practice. 81

V. SUMMARY AND RECOMMENDATIONS. 84

Summary. 84
Recommendations for Further Research . . . 86

Appendices

A. INFORMED CONSENT FORM. 89

B. THERMOMETER AND THERMISTOR SPECIFICATIONS 91

C. DATA COLLECTION SHEETS 93

D. PROTOCOL 97

E. MEAN TEMPERATURE VALUES. 101

REFERENCES. 116

LIST OF TABLES

1. Study Population Characteristics. 47

2. Initial Skin Temperature (^{O} C). 50

3. t-Statistics and p-Values: Initial Skin Temperatures. 51

4. Decrease in Skin Temperature (^{O} C) Following Application of Cold 52

5. t-Statistics and p-Values: Decrease in Skin Temperature. 54

6. Skin Temperatures at Experimental and Control Sites (Phase I) 55

7. Skin Temperatures at Experimental and Control Sites (Phase II). 56

8. Peripheral Blood Circulatory Index Trochanter and Groin. 58

9. Peripheral Blood Circulatory Index Sacrum and Abdomen. 59

10. Two Sample t-test (Unequal Variance) PBCI Male and Female. 60

11. ANOVA of PBCI by Age. 61

12. ANOVA of PBCI by Diastolic Blood Pressure . . . 62

13. ANOVA of PBCI by Height/Weight Ratio. 63

14. Two Sample t-test (Unequal Variance) PBCI Right and Left Trochanter. 64

15. Correlation Matrix: PBCI Experimental Sites. . 65

16. Correlation Coefficients Initial Temperature Decrease versus PBCI. 67

17. Baseline Skin Temperatures (° C): Howell (n=50) and Present Study (n=30). 72

ACKNOWLEDGMENTS

The author extends sincere appreciation to the members of the Supervisory Committee, Mary Ann Johnson, R.N., M.S. and James S. Wood, M.D. for their continued support and encouragement. A special thank-you is extended to Sue E. Huether, R.N., Ph.D., Chairperson of the Supervisory Committee, without whose diligent efforts and technical contributions, this project would not have been possible.

CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Introduction

Pressure ulcers, also known as decubitus ulcers and bed sores, are areas of necrotic tissue caused by the application of prolonged, elevated pressure to the affected area. They are a common nursing problem, and estimates of their incidence in hospitalized patients range from 3.0% (Barbanel, Jordan & Nichol, 1977) to 8.8% (Peterson & Bittman, 1971). The incidence of pressure ulcers in geriatric facilities is probably even higher. One estimate is that 20% of the patients in such a facility will develop a pressure ulcer sometime during their stay (Isiandinso, 1979).

Pressure ulcers are not benign. Once the ulcer has developed, the care of the patient is time-consuming and costly. The average length of hospitalization required for the treatment of pressure ulcers in one spinal paralysis unit was three months (McClemont, Shand & Ramsey, 1977). In 1979, the cost of treating a single pressure ulcer was estimated to be $14,000

(Nola & Vistnes, 1980), with infection, sepsis, and death as the most serious complications. In one series, 48% of all patients with septicemia due to pressure ulcers died (Galpin, Chow, Bayer & Guze, 1976).

Pressure ulcers occur in areas of the body subject to anoxic necrosis. When pressure exceeding capillary intraluminal pressure is applied to the surface of the body, the pressure is transmitted to the capillaries, and capillary flow is interrupted. If the duration and magnitude of pressure application are sufficient, tissue necrosis results (Reuler & Cooney, 1981). The sites of most common occurrence are the tissue overlying the sacrum, ischia, and femoral trochanters (Peterson & Bittman, 1971; Richardson & Meyer, 1981; Roberts & Goldstone, 1979). These areas have also been found to be subject to high pressures in the lying and sitting positions (Kosiak, 1959; Linden, 1961).

Prevention of pressure ulcers is a problem unique to nursing. Other disciplines may investigate the causes of pressure ulcers, and physicians may prescribe programs for their treatment, but it is nurses who must provide the around-the-clock, day-to-day care that is required for their prevention.

Recommendations for nursing interventions aimed

at preventing pressure ulcers generally emphasize the importance of reducing the magnitude and duration of pressure application. These interventions are soundly based on scientific knowledge. However, a number of common nursing interventions are less soundly based on scientific knowledge. For example, massage of bony prominences is frequently mentioned in nursing textbooks as a preventive measure (Beland & Passos, 1975, p. 889; Beyers & Dudas, 1977, p. 1130, Brunner, Emerson, Ferguson & Suddarth, 1970, p. 170; Brunner & Suddarth, 1974, p. 22; Martin, 1971). The use of heat lamps is common in the prevention and treatment of pressure ulcers. A common rationale stated by nurses is that their use increases local circulation, although this has not been documented in the research literature.

The contribution of an impaired baseline local circulation to the development of pressure ulcers is unknown. Little information is available regarding the normal circulation of areas susceptible to pressure ulceration. The most common type of study has utilized measurement of skin temperature to estimate changes in skin blood flow. A reactive hyperemia has been observed following the application of pressure in these areas (Goller, Lewis & McLaughlin, 1971; Mahanty & Roemer, 1979; Verhonick, Lewis & Goller, 1972). The

skin temperature of areas susceptible to pressure ulcers has been found to be lower than that of adjacent areas. However, variations in skin temperature are due to a number of factors other than local blood flow, including the metabolic rate of underlying tissue and the thermal conductivity of that tissue (Mahanty & Roemer, 1980). Since areas susceptible to pressure ulceration consist of a layer of skin with little underlying muscle or subcutaneous tissue, it is possible that the temperature variations observed are due not to variations in local circulation, but to variations in heat production by metabolism. Additional studies utilizing other methods of measuring skin blood flow in areas susceptible to pressure ulceration have yielded incomplete and contradictory evidence (Holloway, Dalt, Kennedy & Chimoskey, 1976; Larsen, Holstein & Lassen, 1979).

If those nursing interventions aimed at preventing pressure ulceration by stimulating local circulation are to be based on scientific knowledge, the circulation of these areas must be studied. Further comparison of these findings with information regarding the local circulation in areas not prone to pressure ulceration would aid in the determination of a scientifically based nursing intervention.

Baptista (1970) has described a simple thermal method for studying skin circulation. In the "thermal recovery method," cold is applied to the area of interest and the rate at which the skin temperature returns to normal is measured. This rate is an index of the circulation of the skin, and has been favorably compared with clearance of radioactive xenon as a method of studying skin circulation (Tavares, Farreta & Fernandes, 1975).

The purpose of this study was to determine if there is a difference between the cutaneous blood flow of areas prone to pressure ulceration, and adjacent, less susceptible areas, as measured by the thermal recovery method.

Review of the Literature

A number of factors including malnutrition, old age, obesity, circulatory deficiency, chronic illness, anemia, blood dyscrasias, skin friability, motor or sensory deficit, shear forces, friction, edema, and pressure have been suggested as contributing factors in the development of pressure ulcers (Argis & Spira, 1979; Griffith, 1963; Isiandinso, 1979; Schell & Wolcott, 1966; Williams, 1972). Only pressure has been consistently shown to cause ulceration, but some evidence suggests that friction (Dinsdale, 1974) and

shear force (Bennett, Kavner, Lee & Trainor, 1979; Palmer, 1979) also play a role in the development of pressure ulcers.

Effects of Pressure on the Etiology of Pressure Ulcers

The vast majority of research on the etiology of pressure ulcers has focused on the effects of externally applied pressure. Experiments have been carried out utilizing isolated tissue and enzymes, unicellular organisms, animals, as well as human subjects.

Isolated tissues and enzymes and unicellular organisms. Cattell (1936) carried out experiments with isolated tissue, enzymes, and unicellular organisms in an attempt to determine if any physiologic effects could be produced as the result of pressure alone. Pressures in excess of 250,000 pounds per square inch were, at times, required to produce changes in cellular function. Since pressure alone cannot be responsible for cellular necrosis, a less direct effect must be responsible. Further research has led to the basic assumption that pressure ulcer formation is due to compression of capillaries by externally applied pressure, resulting in tissue anoxia, deprivation of nutrients, and accumulation of cellular waste products, leading eventually to tissue death (Dinsdale, 1973;

Kosiak, 1961; Trandel, Lewis & Verhonick, 1975).

Animal studies. Because of the obvious ethical problems associated with producing pressure ulcers in human subjects, many studies of the etiology of pressure ulcers utilize animals. Brooks and Duncan (1940) applied pressure to rats' tails and found that the development of pressure ulcers was dependent on both the magnitude and duration of pressure application. This finding has been substantiated by a number of researchers (Daniel, Priest & Wheatley, 1981; Dinsdale, 1973; Kosiak, 1959). Hussain (1953) studied mouse tissue microscopically after the application of pressure, and found changes similar to those in tissues deprived of their blood supply. These findings support the hypothesis that pressure interrupts the blood supply, causing pressure necrosis.

Kosiak (1959) produced pressure ulcers in dogs by both the application of high pressure for short periods of time and application of lower pressure for long periods of time. The time pressure relationship was inverse and followed a parabolic curve. Microscopic changes indicative of anoxic necrosis were found in tissues subjected to as little as 60 mmHg pressure for as short a period of time as one hour.

Kosiak (1961) also applied pressure to the ham-

string muscles of rats, and found a marked susceptibility of tissue to application of moderate pressure for relatively short periods of time. Tissue was less susceptible to alternating pressure than to constant pressure.

In another study (Daniel et al., 1981) pressure was applied over the femoral trochanters of swine. Tissue was found to be more resistant to pressure alone than previously thought, but friction combined with pressure greatly increased the susceptibility of tissue to ischemic necrosis. Muscle was found to be more susceptible to pressure ulceration than skin.

These studies indicate that pressure ulcer formation is dependent on both the magnitude and duration of pressure application, and that relief of pressure for short periods is protective. Although muscle is more susceptible to pressure induced ischemia than skin, pressure ulcers are primarily a problem of the skin, for a number of reasons. Necrosis of underlying muscle may produce no external signs. A force applied to the surface of the body leads to a cone shaped pressure gradient, with the force becoming spread out, and less pressure being applied to deeper layers of tissue (Nola & Vistnew, 1980; Reuler & Cooney, 1981). As long as the skin remains intact and maintains its

function as a barrier, infection even of necrotic underlying tissue is unlikely. However, when the skin is broken, infection, sepsis, and death may result.

Human studies. Studies of the etiology of pressure ulcers in humans have been confined to measurement of capillary pressure, observation of the distribution of pressure over the surface of the body, epidemiological studies, and measurement of skin blood flow. Landis (1930) utilized the microinjection method to determine the capillary pressure in the base at the skin of the fingernail, and found the mean pressure in the arteriolar limb to be 32 mmHg. Other researchers (McClellan, McClellan & Landis, 1942), using pressure plethysmography, found capillary pressure in the forearm to range from 16-33 mmHg. The minimum pressure required to produce pressure ulcers is greater than the capillary pressure, supporting the hypothesis that pressure occludes capillary flow.

Kosiak (1958) measured the pressures under the ischial tuberosities of subjects in the sitting position, and found it to exceed 300 mmHg when the subject was sitting on a flat, unpadded surface. Linden (1961) measured the distribution of pressure over the surface of subjects in the supine, prone, and lateral recumbent positions. In the supine posi-

tion, he found the pressure applied to the sacrum was 60-70 mmHg. In the lateral recumbent position he found pressures of 70-95 mmHg applied to the hip, and in the sitting position, pressures of 65-100 mmHg over the ischial tuberosities.

A number of investigators (Peterson & Bittman, 1971; Richardson & Meyer, 1981; Roberts & Goldstone, 1979) have observed the incidence of pressure ulcers in various parts of the body. They found that pressure ulcers occur most frequently over the sacrum, ischia, and trochanters.

The highest pressures are generated over the sacrum, trochanters, and ischia. These pressures exceed capillary pressure. Pressure ulcers occur most frequently at these sites. Based on these findings, it can be inferred that when pressure is applied to the surface of the body in sufficient magnitude, that pressure is transmitted to the underlying capillaries. If the transmitted pressure exceeds capillary intraluminal pressure, the capillaries collapse, interrupting the blood supply. If this pressure is prolonged, anoxic necrosis of tissue results, forming a pressure ulcer. Interruption of the pressure for short periods is protective, to a degree.

Skin Circulation

Research indicates that pressure ulcers are caused by interruption of capillary blood flow to the skin. However, little information is available regarding the normal or pressure-altered skin blood flow in areas prone to pressure ulceration.

Because of the role of the skin vasculature in blood pressure control and heat regulation, the vasculature of the skin is far more extensive than the nutritional needs alone of the skin would require (Ryan, 1976). The skin is supplied with an extensive network of vessels with great variety and density, and many anastamoses at all levels (Montagna & Prakkal, 1974). This great complexity, as well as variations between individuals, and in the same individual with advancing age has created difficulties in studying skin circulation (Ryan, 1976). Consequently, little information is available regarding the local cutaneous circulation of areas susceptible to pressure ulceration.

Perforator arteries are the primary source of blood supply to the skin. These vessels pass through muscle, give off vessels to the muscles, and continue to the skin. A few direct cutaneous vessels also pass through muscle without giving off branches.

Techniques for Measurement of Skin Circulation

A number of techniques have been utilized in the study of the circulation of the skin. The skin circulation of areas prone to pressure ulceration has been studied by microscopic examination of tissue, clearance of radioactive markers, and various thermal methods. Because so little is known about the circulation of areas prone to pressure ulceration, it is appropriate and necessary to examine the methods used to study skin circulation.

Microscopic examination of the vessels in tissues subjected to a pressure load has been used to observe circulatory changes associated with pressure ulceration. Since this method involves producing a pressure ulcer and then removing a slice of tissue for examination, it is used only in animals. This method also does not provide information about the dynamic changes in skin blood flow that accompany application of pressure.

Two invasive methods are frequently used to study skin blood flow. Systemic injection of fluorescein dye and observation of the resulting fluorescence has been used to study skin circulation (Ryan, 1973). Two days are required for clearance of the dye, and allergic reactions occur. Use of this method to study the skin circulation in relation to the etiology

of pressure ulcers has not been reported in the literature.

Another invasive method involves measuring the rate of clearance of radioactive substances from an intradermal depot. ^{133}Xe and ^{131}I-antipyrine have been injected into the skin and their rate of clearance measured by a radiation detection device in the study of pressure ulcers (Holloway et al., 1976; Larsen et al., 1979). The equipment for this measurement is expensive and inconvenient to use, and special precautions are required for handling of radioactive substances. The intradermal injection may also increase blood flow and thus distort flow values (Holloway, 1980).

A wide variety of noninvasive measurement techniques have been used to study skin blood flow. Plethysmography records changes in volume and requires enclosure of the area of study (Ryan, 1976). This method is not adaptable to study of areas prone to pressure ulceration because the area of interest cannot be isolated.

Laser doppler velocimetry has been used to estimate skin blood flow. The doppler shift produced in a beam of laser light by the movement of red blood cells is measured (Holloway & Watkins, 1977). Although

this instrument is sensitive, reliable and convenient for clinical use, it is expensive and not readily available.

A readily available, inexpensive, and clinically useful method of estimating skin blood flow is measurement of skin temperature. Temperature is the degree of intensity of heat. Although temperature can be qualitatively sensed by receptors in the skin, its quantity cannot actually be measured. Changes in the physical properties of elements such as the volume of a gas, the pressure of a gas, the length of a piece of metal, or the electrical properties of a metal, occur with changes in temperature and can be used to provide quantitative measures of temperature (Mackay, 1970). The only instruments capable of detecting small, rapid changes in skin temperature are electrical thermometers. There are several types of electrical thermometers, including radiation thermometers, resistance thermometers, thermocouples, and thermistors.

Radiation thermometers, also called thermographs, measure a portion of the infrared spectrum emitted by the body surface and transform it into a pictoral display (Verhonick et al., 1972). Thermography is usually used to determine temperature patterns in the skin and not for quantitative measurements of

small, rapid changes in skin temperature.

Thermocouples and thermistors are attached directly to the surface of the skin and are capable of quantitatively measuring small rapid changes in temperature. These probes do not actually measure the temperature of the skin but indirectly reflect the temperature of a probe in contact with the skin. The temperature of the probe must be allowed to equalize with the skin temperature before it can provide an accurate reflection of the skin temperature.

Thermocouples measure the electromotive force generated by the junctions of two dissimilar metals at different temperatures. The probes are suitable for the measurement of core or skin temperature (Stoll & Hardy, 1950).

Thermistors measure temperature changes indirectly by measuring the changes in resistance that occur in heavy metal oxides in proportion to changes in temperature. As the temperature of the thermistor increases, its resistance decreases, following a logarithmic curve. A minute amount of current is passed through the thermistor, and changes in resistance are measured by determining the amount of current passing through a galvanometer. Since the temperature-resistance curve is not linear, the galvanometer used with the thermistor

requires a nonlinear scale. This difficulty can be overcome by constructing a bridge circuit in the instrument to compensate for the nonlinear relationship.

Thermistors have a large change in resistance with relatively small changes in temperature, a rapid response time, and are relatively inexpensive. The disadvantages of thermistors include instability (their resistance gradually increases with age) and the need to recalibrate when thermistors (or thermometers) are changed (Krog, 1962).

Simple measurement of skin temperature has been used frequently to estimate skin blood flow in pressure ulcer research (Barton, 1973; Goller, Lewis, McLaughlin & Verhonick, 1971; Howell, 1981; Mahanty & Roemer, 1979; Newman & Davis, 1981; Trandel, Lewis & Verhonick, 1975; Verhonick et al., 1972). If environmental factors are constant, skin temperature is dependent on the rate of skin blood flow, the temperature of the blood, the metabolic rate (and heat production) of underlying tissue, and the thermal conductivity of that tissue (Mahanty & Roemer, 1980).

In the healthy human, blood temperature does not change significantly during the period required for most blood flow measurements. The thermal conductivity of tissue at a given site is constant. In the resting

subject the metabolic rate of unstressed tissue remains relatively constant. Therefore, observations of temperature changes at the same unstressed site are probably relatively reliable indicators of changes in skin blood flow.

However, simple measurement of skin temperature is a much less reliable method of measuring differences in skin blood flow of different areas of the body. The thermal conductivity and metabolic rate of underlying tissue vary greatly from site to site. The metabolic rate of muscle is higher than that of skin (Nola & Vistnew, 1980). Therefore, the skin temperature of sites with underlying muscle will be warmer than that of sites with little or no underlying muscle.

Thermal Recovery Method

One thermal method of determining skin blood flow that overcomes the problem with simple thermometry has been described by Baptista (1970). In the "thermal recovery method," cold is applied to the skin in the area of interest, and the rate at which the skin returns to its normal temperature is measured. The time required for recovery to the original skin temperature is an index of the peripheral blood flow in the region studied. Baptista described the peripheral blood flow circulatory index (PBCI) as the time re-

quired for the skin temperature to return half way to its original value, and predicted that it would be smaller when the blood flow rate was greater. Since this method is less sensitive to variations in local metabolism than simple thermometry, it is a better measure of local circulation. This method is simple, noninvasive, and inexpensive.

The thermal recovery method has been favorably compared with clearance of radioactive xenon as a method of studying skin circulation (Tavares et al., 1975). Both methods were used to measure the local circulation in the lower legs of three groups of subjects: normal subjects, hyperthyroid subjects with elevated skin temperature, and patients with presumably low cutaneous circulation (hypothyroid patients and those with obstructive atherosclerosis). The mean blood flow values were determined for each group and compared to the mean of the normal group for each method. The significance of difference of means was determined. For the thermal recovery method, the values between normal subjects and those with hypocirculation were: $t=3.36$, $p< 0.001$; between normal subjects and those with hypercirculation: $t=4.04$, $p< 0.0001$. For the xenon clearance method the values were: between normal and hypocirculation: $t=3.98$,

$p < 0.0001$; and between normal and hypercirculation: $t=2.33$, $p < 0.01$. The authors conclude that the thermal recovery method is more sensitive for studying skin hypercirculation, and less sensitive than xenon clearance for studying skin hypocirculation.

The cold applied in the thermal recovery method may cause local vasoconstriction and increase the viscosity of the blood locally (Dodd, 1979). However, because of the relatively short period of time the cold is applied (one minute) these effects are probably minimal. The thermal recovery method is not a method of directly measuring skin blood flow, but measures the skin's ability to respond to the application of cold. Since the ability of the skin to respond to the application of cold is dependent primarily upon its circulatory capacity, the thermal recovery method is a good method of estimating local circulation.

Changes in Skin Circulation Associated with Pressure Ulceration

Some of these methods of investigating skin circulation have been used to determine the changes in circulation that are associated with pressure ulcer formation. These studies have contributed to the understanding of the etiology of pressure ulcers. However, the contribution of baseline differences in

local skin circulation to the development of pressure ulcers is not known.

Some investigators have examined vessels microscopically following the application of a pressure load. Hussain (1953) applied pressure to rats' legs, and described a reactive hyperemia, edema, and frequently capillary and venous hemorrhage in the affected area following the release of pressure. He also showed that it was at this stage that the permeability of vessel walls increased, and localization of particulate matter, such as bacteria, occurred.

Linden (1961) observed rabbits' ears following the application of pressure, and found a reactive hyperemia, followed by edema, dilatation of blood vessels, and extravasation of blood elements. Vascular changes lasted up to 12 days following removal of pressure that did not produce an ulcer.

Dinsdale (1973) produced experimental ulcers in swine, and observed the formation of erythrocytic thrombi, followed by fibrin thrombi in venules and capillaries. He also noted changes in the ultrastructure of the capillaries.

Barton (1973) examined mouse tissue that had been subjected to pressure and found a partial separation of endothelial cells. He felt that trauma could then

cause the cell junctions to break easily, exposing the blood to subendothelial tissue, and causing occlusion of the vessels by platelet thrombi.

These studies suggest that changes in vascular structure resulting from pressure contribute to ulceration by prolonging the period of decreased circulation after pressure is removed. However, other methods of studying skin blood flow are needed to study the dynamic changes that occur when pressure is applied to tissue.

One research group (Larsen et al., 1979) measured the "blood flow cessation external pressure" which is the pressure required to prevent the washout of an intradermal depot of ^{133}-antipyrine, in the sacral and lumbar skin of human subjects. They found that the FCEP correlated strongly with the mean brachial arterial pressure, and was in the range of 63-138 mmHg. These findings could be interpreted to support the assumption that higher external pressures are required to produce pressure ulcers in hypertensive patients. A corollary of this assumption would be that hypotension increases the risk of pressure ulcer formation. This corollary is supported by this investigator's observation that hypotensive patients develop pressure ulcers with high frequency.

Larsen et al. (1979) also found no significant dif-

ference between the FCEP of sacral and lumbar skin. They interpreted this finding as evidence that local circulatory differences are not responsible for pressure ulcer formation, apparently because few pressure ulcers develop in the lumbar region. The sacral and lumbar regions both have relatively thin layers of muscle and subcutaneous fat between skin and underlying bone. Since the vasculature of the skin is primarily derived from the underlying muscle, it is likely that the skin vasculature of these areas is similar. An area with substantial layers of intervening muscle would likely have a different skin vasculature. Comparison of the skin circulation of an area prone to pressure ulceration with little underlying muscle with an area with substantial underlying muscle would probably yield more valuable information regarding the contribution of local differences in circulation to the development of pressure ulcers.

Another group of researchers (Holloway et al., 1976) utilized clearance of Xe^{133} to compare the skin circulation of the sacrum and forearm. They found that lower pressures were required to occlude capillary flow in sacral skin than in forearm skin. These findings could be used to support the assumption that local skin circulatory differences do contribute to the

development of pressure ulcers. However, the forearm and sacrum are widely separated areas, and factors other than local circulatory differences could intervene. It is also probable that the varying amounts of padding between the two areas cause greater pressures to be transmitted to the capillaries of the sacral skin. These studies do not measure the baseline circulation, but the ability of the circulation to function under a pressure load. Studies using clearance of radioactive substances to study skin circulation of areas prone to pressure ulceration are incomplete and contradictory.

A number of studies have utilized skin temperature measurement to estimate skin blood flow in relation to pressure ulcer formation. In one study (Goller et al., 1976) pressure was applied to human skin and the skin temperature was measured with thermography following the release of pressure. An increase in temperature was noted following release of pressure.

In another study (Verhonick et al., 1972) pressure was applied to the body surface by having the subject lie on the area to be studied, and skin temperature was measured thermographically after pressure was removed. An initial cooling at the highest pressure point was noted, followed by a "flushing" to a temper-

ature above normal, and then a return to the normal temperature distribution.

Mahanty and Roemer (1979) applied pressure over the femoral trochanters of six young human subjects and measured the skin temperature with thermocouples after the pressure was released. They found an increase in skin temperature that was directly proportional to the amount of pressure and the length of time it was applied.

Newman and Davis (1981) obtained thermographs of the ischial and sacral areas of hospitalized geriatric patients after a load had been applied by having the patient lie on the area to be studied. They found that patients who demonstrated a "thermal flare" after pressure had been released were more prone to pressure ulcer formation.

The initial cooling observed by Verhonick et al. is probably due to evaporative cooling. Moisture on the surface of the body would not evaporate while the subject was lying on the area of study. When the area was exposed for thermography, evaporative cooling could occur.

The increase in temperature observed following release of pressure can be explained by the mechanism of reactive hyperemia. After blood flow is occluded

by pressure, and then reestablished by the release of pressure, circulation to the area is increased as a compensatory mechanism. Lewis and Grant (1925) and Goldblatt (1926) studied reactive hyperemia in man, and found that it was a local reaction, independent of central nervous system and hormonal control. It occurred whenever the arterial supply was occluded, and was characterized by vasodilatation. The extent and duration of the reactive hyperemia has been found to be proportional to the local metabolic debt (Lewis & Landis, 1929). By this mechanism, tissue temporarily deprived of its blood supply is protected from necrosis.

The skin temperature of subjects lying on a transparent surface was measured in another study (Trandell et al., 1975) by obtaining thermographs of the subjects taken through the transparent surface. Areas prone to pressure ulceration, which were also exposed to the highest pressures, were found to have the highest skin temperature. One would expect that the areas subjected to the highest pressure would have decreased skin blood circulation and lower skin temperatures. The authors offered no explanation for this seemingly contradictory finding. Since the areas that were subjected to the highest pressures were in closest contact with the transparent surface, heat loss in

these areas was probably minimal. It is likely that in this study, the increased skin temperature was not due to increased circulation but rather to decreased heat loss.

Howell (1981) measured the skin temperature of elderly, ambulatory, ulcer-free females over the trochanters, sacrum, heels, and lateral malleoli, and compared these temperatures to the skin temperature of adjacent areas not prone to pressure ulceration. He found that areas prone to pressure ulceration were cooler than the adjacent areas. Howell explained the cooler skin temperatures of areas prone to pressure ulceration as being due to decreased circulation in these areas. However, areas prone to pressure ulceration are characterized by very little or no underlying muscle or bone (Nola & Vistnew, 1980). Therefore, the differences in temperature may be due to increased heat production by muscle in the less susceptible areas rather than differences in skin circulation.

These studies of the local circulation in relation to pressure ulcer formation showed that changes in the ultrastructure of the vasculature occur following application of pressure, at least in animal studies. A reactive hyperemia has been shown to occur following the release of pressure. Very little research has

been conducted to study the baseline circulation of areas prone to pressure ulceration. Since the skin vasculature is derived from underlying muscle, areas prone to pressure ulceration may have a decreased circulation. The evidence of whether circulation in these areas is decreased is incomplete and contradictory.

Summary

Because of the obvious ethical problems of deliberately producing pressure ulcers in human subjects, much of the basic research on pressure ulcers has been done with animals. Animal studies have shown that pressure ulcers are produced by externally applied pressure which causes occlusion of the capillaries, and results in tissue necrosis. The development of pressure ulcers has been shown to be dependent on both the magnitude and duration of pressure application. Microscopic changes in the vasculature of skin have been observed following the application of pressure. Human studies have shown that pressure ulcers develop most commonly in areas that are subject to the highest pressure, primarily the sacrum, hips, and ischia.

Little information regarding the local circulation of areas susceptible to pressure ulceration is avail-

able. Two studies utilizing the clearance of radioactive substances showed that skin blood flow ceases at pressures thought to be capable of producing pressure ulcers, but the data is incomplete and contradictory. Studies utilizing thermometry as a means of studying local skin circulation in these areas have recently become quite popular. These studies have shown that areas prone to pressure ulcer formation demonstrate increased temperature after a pressure load is applied. Other skin temperature studies have shown that areas prone to pressure ulcer development are cooler than adjacent areas. Since skin temperature is dependent on metabolic rate as well as the rate of blood flow, the question of whether decreased baseline local blood flow contributes to the development of pressure ulcers has not been answered.

The thermal recovery method is a simple, noninvasive method of estimating local skin blood flow, and is less sensitive to metabolic differences than simple thermometry. Thermistors are an appropriate means of measuring skin temperature.

CHAPTER II

CONCEPTUAL FRAMEWORK

Assumptions

The following assumptions were used in formulating a conceptual framework for this study:

1. Pressure ulcers are areas of hypoxemic necrosis caused by compression of capillaries by externally applied pressure.

2. If the skin blood flow is decreased, a less severe pressure load is required to compress the capillaries and cause pressure ulceration.

3. The skin blood flow varies from one area of the body surface to another. Decreased skin blood flow may be a contributing factor to the high incidence of pressure ulceration in certain areas of the body.

4. The rate at which cooled tissue is rewarmed is a function of local skin blood flow.

Conceptual Framework

Pressure ulceration is due to compression of the capillaries by externally applied pressure, resulting

in interruption of blood flow and hypoxemic necrosis. When the pressure applied to the capillaries exceeds capillary intraluminal pressure, the capillaries are compressed and blood flow decreases. If capillary intraluminal pressure is decreased, less external pressure is required to interrupt blood flow. Capillary pressure is determined by flow and resistance. When the flow through the capillaries is decreased, capillary intraluminal pressure is decreased. Thus, when skin blood flow is decreased, a less severe pressure load is required to interrupt flow, and the risk of pressure ulceration is increased.

Skin blood flow in certain areas of the body may be less than in other areas. These areas would be at greater risk of developing pressure ulcers. Pressure ulcers develop most frequently over the ischia, trochanters, and sacrum. The sacrum and trochanters have very little muscle between skin and underlying bone. The vasculature of the skin is derived from underlying muscle. Therefore, the skin blood flow in these areas may be decreased. This decreased blood flow would contribute to the development of pressure ulcers in these areas. Determination of any differences between skin blood flow of these areas and other areas not prone to pressure ulceration, will contribute to

understanding of the etiology of pressure ulceration.

A number of techniques exist for measuring skin blood flow. Most of these techniques involve the use of expensive equipment that is not readily available. Measurement of skin temperature is a technique for estimating skin blood flow that requires less-expensive, more readily-available equipment. However, skin temperature is affected by factors other than blood flow, including metabolic rate of underlying tissue.

If cold is applied to the surface of the body, heat moves from the warmer area (the body) to the cooler area (the cold applicator). After the cold applicator is removed, the area to which it was applied is cooler than the surrounding tissue. Heat again moves from the warmer area to the cooler area.

Blood is warmer than the cooled area. If a large amount of blood moves quickly to the cooled area, heat will be transferred more quickly than if a small amount of blood moves sluggishly to the cooled area. Therefore, the time required for thermal recovery of tissue is a function of the circulatory capacity of the tissue.

The skin circulation can be estimated by applying cold to the skin and measuring the rate at which its temperature returns to the original value. A more

rapid recovery indicates that the skin blood flow is greater, while a slower recovery indicates that the skin blood flow is less.

The purpose of this study was to determine if the skin blood flow, as measured by the thermal recovery method, is decreased in areas prone to pressure ulceration such as the sacrum and trochanters, when compared with adjacent, nonsusceptible areas.

Research Questions

The specific research questions to be answered in this study were:

1. Is the peripheral blood flow circulatory index (PBCI) over the trochanter significantly higher than the PBCI over the groin?

2. Is the PBCI over the sacrum significantly higher than the PBCI over the abdomen?

Operational Definitions

The following operational definitions were utilized in the study:

1. Peripheral blood flow circulatory index: The time required for the skin temperature to return half way to its original value following the application of cold.

2. Trochanter: The area of body surface des-

cribed by a circle ten centimeters in diameter, with its center over the palpated femoral trochanter.

3. Groin: The area of body surface described by a circle ten centimeters in diameter, with its center midway between the anterior superior iliac spine and the symphysis pubis.

4. Sacrum: The area of body surface described by a circle ten centimeters in diameter, with its center over the most prominent palpated sacral tubercle.

5. Abdomen: The area of body surface described by a circle ten centimeters in diameter, with its center midway between the xyphoid process and the umbilicus.

6. Right brachial region: The area of body surface described by a circle ten centimeters in diameter centered over the biceps muscle on the right arm.

7. Experimental sites: A randomly selected trochanter, the ipsilateral groin, the sacrum, and the abdomen.

8. Control sites: The contralateral groin and the right brachial region.

9. Skin temperature: Expressed in degrees centigrade, measured by skin thermistors attached to the sites of interest, and displayed on an electronic thermometer.

CHAPTER III

METHODOLOGY AND RESEARCH DESIGN

Aim

The aim of this study was to determine if skin blood flow, as measured by the thermal recovery method, is less in areas susceptible to pressure ulcers than in adjacent, nonsusceptible areas. Skin circulation was measured over the trochanter and sacrum, and compared with the skin circulation measured over the groin and abdomen. This information will contribute to understanding of the etiology of pressure ulcers and ultimately to determining appropriate nursing care for their prevention and treatment.

Population

The study population consisted of a convenience sample of 30 subjects selected from a geriatric health screening center staffed by health science students from the University of Utah. The center offered a number of services including blood pressure screening, hematocrit and blood glucose testing, nutritional

counseling, pedicures, cerumen removal, and physical examinations. Subjects were recruited from clients utilizing these services.

Subjects were approached in the waiting area and after a brief explanation, were asked if they would be willing to participate in the study. Subjects who met the following criteria were asked to participate in the study:

1. At least 60 years of age.
2. Willing to sign the consent form.
3. Willing to cooperate with the measurement procedures for about 45 minutes.
4. No history of diabetes mellitus or serious skin disorders.
5. Lack of current pressure ulcers or skin disorders.

Design

The design of this study was descriptive. All thermal recovery measurements were made under similar conditions on all subjects. Each subject served as his/her own control.

Instruments

All instruments used for data collection were noninvasive. All measurements were made by the investi-

gator.

Thermometers

Two Digitec electronic thermometers with multiple monitoring channels (Model 5810) and Yellow Springs Instrument thermistor button probes were used to measure skin temperature. The thermistors were held in place on the skin with paper tape and were insulated with a cotton ball (Appendix B details instrument specifications).

Cold Applicators

Two glass containers, each with a diameter of ten centimeters and filled with a slurry of ice and water, were used to apply cold to the skin. A plastic wrap was placed over the opening of the container and held in place with an elastic band to prevent spillage.

Skin Marker

A water soluble marking pen was used to mark the skin for application of cold and placement of thermistors. A glass container identical to those used to apply cold was used as a template to mark circles ten centimeters in diameter.

Sphygmomanometer and Scales

Blood pressure was measured from the right arm

with a mercury sphygmomanometer and a stethoscope, using the standard procedure (Kirkendall, Burton, Epstein & Freis, 1967). Weight was measured on a balance scale. Height was measured with a ruler attached to the scale.

Room Temperature Thermometer

Room temperature was measured before and after thermal recovery measurements on each subject, using a mercury-in-glass thermometer (Springfield No. 096).

Data Collection Procedures

Assignment of Experimental and Control Sites

Because of the pressure load created by the subject lying on an area of study is known to alter skin blood flow, areas not subjected to a pressure load were studied. Patients were randomly assigned to two groups of equal size. In one group of 15, subjects were asked to lie on their left side, and the right trochanter and groin were studied. In the other group of 15 subjects, they were asked to lie on their right side, and the left trochanter and groin were studied.

The study occurred in two phases. During the first phase, the thermal recovery method was used to study the exposed trochanter and the ipsilateral groin. To control for changes in skin temperature due to

environment, measurement, or physiologic factors, skin temperature was recorded at 20-second intervals over the contralateral groin and right brachial region.

During the second phase of the study, the thermal recovery method was used to study the sacrum and abdomen. Skin temperature was measured over the right brachial region at 20-second intervals to serve as a control during the second phase of the study.

Data Recording

Data were recorded by hand on a data collection sheet (Appendix C). Skin temperature was recorded at two experimental (trochanter, ipsilateral groin) and two control (contralateral groin, right brachial region) sites during the first phase of the study. Skin temperatures were recorded at two experimental (sacrum and abdomen) and one control site (right brachial region) during the second phase of the study (Figure 1). Two electronic thermometers, each with three channels were used, and manual switching was required. The same thermistors and thermometer channels were used at each site.

Baseline skin temperature was recorded at all sites five minutes after application of the thermistors. The cold glass container was then applied to the experimental sites for one minute. Following appli-

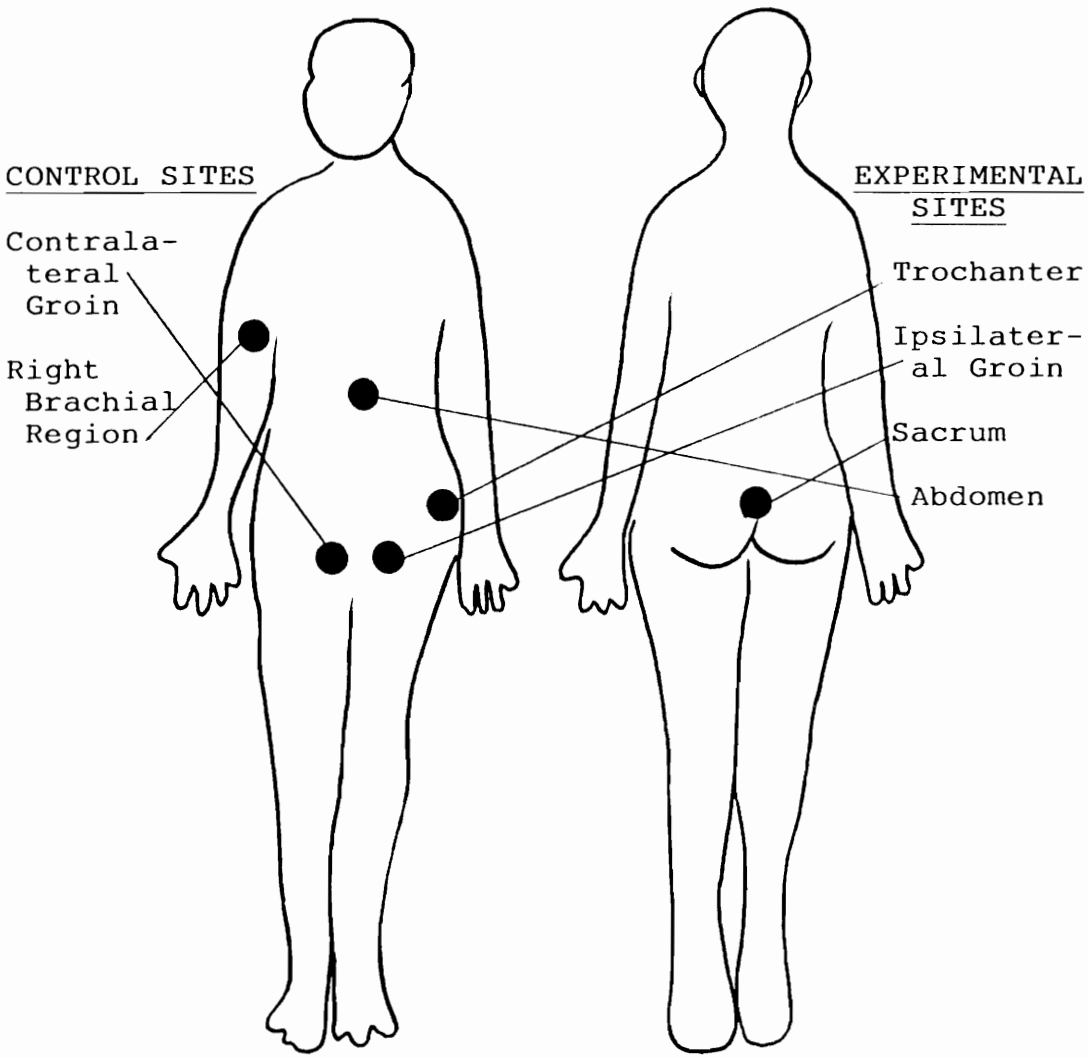

Figure 1. Experimental and control sites.

cation of cold, skin temperature was recorded at 20-second intervals, as determined by the sweep hand on a wristwatch. Skin temperature measurements were recorded until the skin temperature had returned halfway to the baseline skin temperature.

Determination of the Peripheral Blood Flow Circulatory Index (PBCI)

The PBCI is the index of skin circulation of the thermal recovery method. It is the time required for the skin temperature to return halfway to the baseline temperature following the application of cold. In order to calculate the PBCI the following procedure was used:

1. Subtract the lowest skin temperature following the application of cold (T_c) from the baseline skin temperature (T_b). This value is the initial decrease in skin temperature.

2. Divide the initial decrease in skin temperature by two.

3. Add the resulting figure to the lowest skin temperature following the application of cold (T_c). This value is the temperature midway between the baseline skin temperature (T_b) and the lowest skin temperature following the application of cold (T_c) and is termed the calculated $T_{½}$. The formula for calcu-

lation of the calculated $T\frac{1}{2}$ is:

$$T\frac{1}{2} = T_c + \frac{T_b - T_c}{2}$$

4. Compare the calculated $T\frac{1}{2}$ to the recorded skin temperature values. Since skin temperatures were recorded at 20-second intervals, the calculated $T\frac{1}{2}$ seldom corresponds exactly to recorded values. If the calculated $T\frac{1}{2}$ is between two recorded values, the time at which the lower of the two recorded values was measured is the PBCI. Therefore, the accuracy of this measurement is + 19 seconds.

Overview of Data Collected

Demographic data including sex, age, weight, height, blood pressure, medications taken, and medical history were collected on each subject. Room temperature was recorded at the beginning and end of the data collection period.

During the first phase of the study, a baseline temperature was recorded for the experimental sites (exposed trochanter and ipsilateral groin) and the control sites (contralateral groin and right brachial region). Skin temperatures of these four sites were recorded immediately following the application of cold to the trochanter and ipsilateral groin, and at 20-second intervals thereafter. Skin temperatures continued to

be recorded until skin temperature had returned half - way to the baseline temperature.

During the second phase of the study, baseline skin temperatures were recorded at the experimental sites (sacrum and abdomen) and at the control site (right brachial region). Following application of cold to the sacrum and abdomen, skin temperature measurements were recorded at all three sites, using the same procedure as during the first phase of the study (Appendix D lists the complete protocol).

CHAPTER IV

DATA ANALYSIS

Methods of Data Analysis

Descriptive statistics about the mean, the paired t-test, one-way analysis of variance, and the correlation coefficient were used to analyze the data. Calculations were completed using the Tektronix 4052 computer and statistical software.

Descriptive Statistics

The mean, standard deviation, and range were calculated for the following data:

1. The initial skin temperature at each experimental site (Phase one: trochanter and ipsilateral groin; Phase two: sacrum and abdomen) and each control site (Phase one: contralateral groin and right brachial; phase two: right brachial).

2. Skin temperature at each experimental and control site for each 20-second measurement period following application of cold (descriptive statistics for skin temperature are listed in Appendix E).

3. The PBCI for each experimental site (Phase

one: trochanter and ipsilateral groin; Phase two: sacrum and abdomen) for all subjects.

Paired t-Test

The t-test is the basic parametric procedure for testing differences between two group means. Since a single group of subjects was used, and subjects served as their own controls, the paired t-test was used. This procedure is used for testing difference in the means of dependent groups (Polit & Hungler, 1978, pp. 548-551).

The paired t-test was used to test for statistical differences between:

1. The initial skin temperature at all seven sites.
2. The initial decrease in skin temperature of the experimental sites.
3. The temperature of the control sites at the beginning and end of each phase of the study.
4. The PBCIs of the experimental sites.

The t-test for independent variables was used to test for statistical difference between:

1. The PBCIs of the left and right trochanters.
2. The PBCIs of male and female subjects.

Because a directional hypothesis was proposed, a one-tailed t-test was used to determine the p values

between the PBCIs of the trochanter and groin, and between the PBCIs of the sacrum and abdomen. The two tailed t-test was used in all other instances.

Analysis of Variance

Analysis of variance (ANOVA) is a parametric procedure used to test the significance of difference between group means of more than two groups (Polit & Hungler, 1978, pp. 553-559). One-way, completely randomized, analysis of variance was used to test for statistical differences based on:

1. Subject age.
2. Height/weight ratio.
3. Diastolic blood pressure.

Correlation Coefficients

The most common method of describing statistical relationships between two measurements is the correlational procedure (Polit & Hungler, 1978, pp. 528-533). This procedure was used to determine correlational relationships between:

1. The initial decrease in temperature and the PBCI at each site.
2. The PBCIs of all four sites.

Results of Data Analysis

The Study Population

A convenience sample of 30 subjects was obtained. All subjects were clients of the health screening center. Since these subjects were all relatively healthy, ambulatory, living at home, and relatively well-informed and concerned about their health, they were probably representative of the majority of the elderly population.

Sixteen females and 14 males participated in the study. The age of the subjects ranged from 60 to 87 years, with a mean age of 70.6 years (S.D. $\pm$ 7.2). Twelve of the subjects were taking antihypertensive medications. Only two subjects had a diastolic blood pressure higher than 90 mmHg. The right trochanter and groin were subjected to the thermal recovery method in 15 subjects, and the left trochanter and groin were used in 15 subjects. One subject had a pressure ulcer during a hospitalization 20 years previously (Table 1).

Environmental Conditions

The study was conducted from May 10 to June 7, 1983. Measurement took place between one pm and four-thirty pm. Measurements on all subjects occurred in the same small (6x8 feet), unventilated room. During

Table 1

Study Population Characteristics

Subj.	Sex	Age	Blood Pressure	Weight (kg)	Height (cm)	Weight Height Ratio (kg/cm)	Trochanter Studied
1	M	62	134/74	80	173	0.46	R
2	M	68	140/84	57.2	163	0.35	R
3	M	75	128/68	68.2	170	0.40	R
4	M	67	150/84	56.8	168	0.34	R
5	F	85	136/84	56.8	163	0.35	L
6	M	77	134/76	80	179	0.45	L
7	M	68	144/74	75	165	0.45	R
8	F	75	124/76	56.4	163	0.35	L
9	F	87	140/80	55.9	165	0.34	L
10	M	76	124/70	71.8	180	0.40	R
11	M	76	154/90	76.8	174	0.44	L
12	F	78	130/70	56.4	145	0.39	R
13	M	68	140/100	85	178	0.48	R
14	F	70	140/80	83.4	171	0.49	R
15	F	60	126/70	74.5	165	0.45	L
16	M	80	110/50	76.4	173	0.44	L

Table 1 (Continued)

Subj.	Sex	Age	Blood Pressure	Weight (kg)	Height (cm)	Weight Height Ratio (kg/cm)	Trochanter Studied
17	M	72	134/80	65	170	0.38	R
18	F	65	150/84	61.8	161	0.38	L
19	M	72	116/60	73.4	168	0.44	L
20	F	65	118/70	52.3	165	0.32	L
21	F	60	138/78	69.5	169	0.41	R
22	M	70	134/86	73.6	170	0.43	R
23	F	69	142/90	70.2	163	0.43	L
24	F	72	112/60	66.8	160	0.42	R
25	F	64	176/92	69.1	169	0.41	L
26	M	68	138/72	66.8	168	0.40	L
27	F	67	116/60	62.5	164	0.38	R
28	F	62	148/92	70.9	160	0.44	R
29	F	81	150/90	63.6	157	0.41	L
30	F	60	124/78	61.4	173	0.35	L
Mean	F=16	70.6	135/77	67.9	167	0.41	L=15
	M=14						R=15
S.D.		7.2	14.5/11.3	8.9	7.1		

the study, room temperatures ranged from 24° C to 26° C, but did not vary during the course of measurement of any one subject. Subjects wore hospital gowns and their underclothes. Subjects who wore LDS garments were asked to remove them. Subjects were covered with a sheet, and all stated they were comfortable in regard to temperature. None of the subjects shivered during the measurements.

Skin Temperatures

The baseline skin temperatures ranged from 29.5° C to 36.0° C (Table 2). The groins were the warmest areas. The groins were significantly warmer than the trochanter, sacrum, and abdomen. The trochanter was the coolest area. It was significantly cooler than the groin, sacrum, and right brachial regions (Table 3). The mean skin temperatures of the sacrum and abdomen were identical.

Following application of cold, an immediate decrease in the skin temperature of the experimental sites (trochanter, groin, sacrum, and abdomen) was noted (Table 4). Skin temperatures frequently continued to decrease for one to four additional measurement periods (20-80 seconds).

The skin temperature of the sacrum decreased significantly less than that of the trochanter

Table 2

Initial Skin Temperature (^{o} C)

Site	Mean	Standard Deviation	Range
Phase One			
Trochanter	32.7	1.28	29.4-36.0
Ipsilateral Groin	33.9	0.96	31.0-35.0
Contralateral Groin	33.9	1.16	31.4-35.5
Right Brachial	33.2	1.16	31.7-35.8
Phase Two			
Sacrum	33.3	1.22	30.6-35.3
Abdomen	33.3	1.21	30.6-35.3
Right Brachial	33.9	1.12	31.1-35.3

Table 3

t-Statistics and *p*-Values: Initial Skin Temperatures

	Trochanter	Ipsilateral Groin	Contralateral Groin	Brachial #1	Sacrum	Abdomen
Ipsilateral Groin	*t*=-6.252024 *p*=0.0000					
Contralateral Groin	*t*=-5.87805 *p*=0.0000	*t*=.61380 *p*=.54410				
Right Brachial #1	*t*=2.07603 *p*=.04688	*t*=4.08197 *p*=.00032	*t*=3.72367 *p*=.00084			
Sacrum	*t*=2.90454 *p*=.00698	*t*=3.92976 *p*=.00048	*t*=3.10304 *p*=.00426	*t*=0.32656 *p*=.74632		
Abdomen	*t*=1.82247 *p*=.07872	*t*=3.43787 *p*=.00180	*t*=2.83449 *p*=.00828	*t*=.09639 *p*=.92386	*t*=.52788 *p*=.60158	
Right Brachial #2	*t*=-3.52534 *p*=.00142	*t*=.43882 *p*=.66402	*t*=.01654 *p*=.98692	*t*=2.25260 *p*=.03204	*t*=2.18199 *p*=.03736	*t*=2.31331 *p*=.02800

Table 4

Decrease in Skin Temperature (^{O} C)

Following Application of Cold

Site	Mean	Standard Deviation	Range
Trochanter	5.4	1.15	2.5-8.3
Groin	4.8	1.67	0.5-7.7
Sacrum	4.0	1.71	1.6-8.8
Abdomen	4.8	1.51	1.9-7.6

($p < 0.005$) and the abdomen ($p < 0.05$). The skin temperature of the abdomen also decreased less than the temperature of the groin, but the difference only approached significance ($p=.053$). No significant differences were found between the temperature decreases over the trochanter, groin, and abdomen (Table 5).

During the first phase of the study, while the thermal recovery method was being applied to the trochanter and groin, skin temperatures were recorded over the contralateral groin and the right brachial region as a control (Table 6). During phase one the mean skin temperature of the contralateral groin increased 0.3° C ($p < 0.001$). The mean temperature of the right brachial region increased 0.59° C ($p < 0.001$).

During the second phase of the study, while the thermal recovery method was being applied to the sacrum and abdomen, the skin temperature of the right brachial region was recorded as a control (Table 7). During phase two, the mean skin temperature of the right brachial region decreased 0.08° C ($p=0.33$).

Peripheral Blood Circulatory Index (PBCI)

The PBCI data were analyzed in relation to the research questions:

Table 5

t-Statistics and *p*-Values: Decrease in Skin Temperature

	Trochanter	Groin	Sacrum
Groin	*t*=1.47929 *p*=.14984		
Sacrum	*t*=3.60812 *p*=.00114	*t*=2.01748 *p*=.05300	
Abdomen	*t*=1.69256 *p*=.10128	*t*=.20926 *p*=.83578	*t*=2.23643 *p*=.03318

Table 6

Skin Temperatures at Experimental and Control Sites

(Phase I)

	Trochanter	Right Brachial (Control Site)	Groin	Contralateral Groin (Control Site)
Baseline				
Mean	32.7	33.2	33.9	33.9
S.D.	1.28	1.34	0.96	1.16
After Cold				
Mean	27.4	33.5	29.1	34.0
S.D.	1.34	1.14	1.55	1.14
Change in Skin Temperature Between Baseline and Removal of Cold				
Mean	-5.3	+0.3	-4.8	+0.1
S.D.	1.15	0.06	1.67	0.03
p	0.0000	0.002	0.0000	0.001
Baseline				
Mean	32.7	33.2	33.9	33.9
S.D.	1.28	1.34	0.96	1.16
At PBCI Time				
Mean	30.0	33.8	31.4	34.2
S.D.	1.15	1.19	0.96	1.14
Change in Skin Temperature Between Baseline and PBCI Time				
Mean	-2.7	+0.6	-2.5	+0.3
S.D.	0.67	0.1	0.58	0.1
p	0.0000	0.0004	0.0000	0.0002

Table 7

Skin Temperatures at Experimental and Control Sites

(Phase II)

	Sacrum	Right Brachial (Control)	Abdomen
Baseline			
Mean	33.3	33.9	33.3
S.D.	1.22	1.12	1.28
After Cold			
Mean	29.8	33.7	28.7
S.D.	1.82	1.24	1.86
Change in Temperature Between Baseline and Application of Cold			
Mean	-4.0	-0.2	-4.6
S.D.	1.7	0.03	1.5
p	0.0000	0.29	0.00000
Baseline			
Mean	33.3	33.9	33.3
S.D.	1.22	1.12	1.28
At PBCI Time			
Mean	31.3	33.8	30.9
S.D.	1.28	1.24	1.37
Change in Temperature Between Baseline and PBCI Time			
Mean	-2.0	-0.1	-2.4
S.D.	0.8	0.02	0.7
p	0.0000	0.33	0.0000

1. Is the PBCI greater over the trochanter than over the groin?

The mean PBCI over the trochanter was 277 seconds (S.D. ± 93), and the mean PBCI over the groin was 261 seconds (S.D. ± 81). This difference was not statistically significant ($p=.23$) (Table 8).

2. Is the PBCI greater over the sacrum than over the abdomen?

The mean PBCI over the sacrum was 327 seconds (S.D. ± 156), while the mean PBCI over the abdomen was 308 seconds (S.D. ± 144). This difference was not statistically significant ($p=.30$) (Table 9).

Further analysis of the data showed no significant differences in the PBCIs of any sites based on sex (Table 10), age (Table 11), diastolic blood pressure (Table 12), or height/weight ratio (Table 13). No significant differences were found between the PBCIs of the right and left trochanters (Table 14).

Correlation coefficients were calculated between the PBCIs of all the experimental sites (Table 15). A positive correlation was noted between the PBCI of the trochanter and the PBCI of the abdomen ($p < 0.001$). The positive correlation between the PBCI of the sacrum and the PBCI of the groin approached significance ($p < 0.1$).

Correlation coefficients were calculated between

Table 8

Peripheral Blood Circulatory Index

Trochanter and Groin

	Mean	Standard Deviation	Range	p
Trochanter	277	93	120-560	0.23
Groin	261	81	80-800	

Table 9

Peripheral Blood Circulatory Index

Sacrum and Abdomen

	Mean	Standard Deviation	Range	p
Sacrum	327	156	80-800	0.30
Abdomen	308	144	100-780	

Table 10

Two Sample *t*-test (Unequal Variance)

PBCI Male and Female

	Male Mean (*n*=14)	Female Mean (*n*=16)	*t*-statistic	*p*-value
Trochanter	296	264	0.91	0.38
Groin	257	265	0.86	0.80
Sacrum	364	316	1.17	0.41
Abdomen	281	345		0.25

Table 11

ANOVA of PBCI by Age

Site	Source	Sum of Squares	df	Mean Square	F	Significance Level
Trochanter	Between Groups	730.000	2	365.000	0.039	0.963
	Among Groups	251056.667	27	9298.395		
	Total	251786.667	29			
Groin	Between Groups	8830.000	2	4415.000	0.649	0.527
	Among Groups	183556.667	27	6798.395		
	Total	192386.667	29			
Sacrum	Between Groups	93926.667	2	46963.333	2.083	0.142
	Among Groups	608860.000	27	22550.370		
	Total	702786.667	29			
Abdomen	Between Groups	51420.000	2	25710.000	1.268	0.294
	Among Groups	547460.000	27	20276.296		
	Total	598880.000	29			

Table 12

ANOVA of PBCI by Diastolic Blood Pressure

Site	Source	Sum of Squares	df	Mean Square	F	Significance Level
Trochanter	Between Groups	9266.667	3	3088.889	0.331	0.803
	Within Groups	242520.000	26	9327.692		
	Total	251786.667	29			
Groin	Between Groups	4386.667	3	1462.222	0.202	0.894
	Within Groups	188000.000	26	7230.769		
	Total	192386.667	29			
Sacrum	Between Groups	65906.667	3	21968.889	0.897	0.455
	Within Groups	636880.000	26	24495.385		
	Total	702786.667	29			
Abdomen	Between Groups	14760.000	3	4920.000	0.219	0.882
	Within Groups	584120.000	26	22466.154		
	Total	598880.000	29			

Table 13

ANOVA of PBCI by Height/Weight Ratio

Site	Source	Sum of Squares	df	Mean Square	F	Significance Level
Trochanter	Between Groups	23895.758	3	7965.253	0.909	0.449
	Within Groups	227890.909	26	8765.035		
	Total	251786.667	29			
Groin	Between Groups	7355.152	3	2451.717	0.345	0.793
	Within Groups	185031.515	26	7116.597		
	Total	192386.667	29			
Sacrum	Between Groups	51710.606	3	17236.869	0.688	0.566
	Within Groups	651076.061	26	2504.387		
	Total	702786.667	29			
Abdomen	Between Groups	36775.758	3	12258.586	0.567	0.641
	Within Groups	562104.242	26	21619.394		
	Total	598880.000	29			

Table 14

Two Sample t-test (Unequal Variance)

PBCI Right and Left Trochanter

Mean Right Trochanter	Mean Left Trochanter	t-statistic	p-value
269	288	0.54	0.59

Table 15

Correlation Matrix: PBCI Experimental Sites

	PBCI Trochanter	PBCI Ipsilateral Groin	PBCI Sacrum	Abdomen
PBCI Trochanter	1.00000	0.06749	0.20774	0.61455
PBCI Ipsilateral Groin	0.06749	1.00000	0.31290	-0.15131
PBCI Sacrum	0.20774	0.31290	1.00000	0.13540
PBCI Abdomen	0.61455	-0.15131	0.13540	1.00000

the initial decrease in skin temperature and the PBCI at each site. A positive correlation was found between the initial decrease in skin temperature of the sacrum and the PBCI of the sacrum ($p < 0.02$) (Table 16).

Discussion

Research has shown that pressure ulcers are caused by hypoxemic necrosis due to interruption of blood flow to the affected tissue. Areas that are frequently subjected to high pressure, such as the sacrum and trochanter, develop pressure ulcers with high frequency. Research conducted on the skin circulation of these two areas has shown that blood flow decreases during the application of pressure, and that a reactive hyperemia occurs following the release of pressure (Barton, 1973; Goller et al., 1976; Kosiak, 1959; Nola & Vistnes, 1980). The contribution of local differences in the baseline circulation of skin that has not been subjected to a pressure load to the development of pressure ulcers is unknown.

In order to determine the contribution of local differences in skin circulation to the development of pressure ulcers, this study addresses two research questions:

1. Is the peripheral blood flow circulatory index (PBCI) of the trochanter significantly greater than the PBCI

Table 16

Correlation Coefficients Initial

Temperature Decrease versus PBCI

Trochanter	-0.30
Groin	0.29
Sacrum	0.45
Abdomen	0.27

of the groin?

No significant differences were found between the PBCIs of these two sites. Since the PBCI reflects the rates of blood flow in these two areas, it is concluded that blood flow comparisons, as measured by the thermal recovery method, between the trochanter and the groin do not differ significantly.

These findings are contradictory to earlier interpretations (Barton, 1973; Howell, 1981; Trandel et al., 1975) of skin temperature values. These researchers have assumed that the decreased skin temperature of areas prone to pressure ulceration is due to decreased blood flow in these areas. The results of the current study suggest that these skin temperature differences are not due to differences in skin blood flow. Areas prone to pressure ulceration usually consist of skin with relatively thin layers of underlying muscle or subcutaneous fat. Since muscle is metabolically more active than skin, it produces more heat than skin. It is probable that areas prone to pressure ulceration are cooler than other areas because less heat is produced by underlying tissue in these areas.

This investigation speculated that areas of the body with decreased skin circulation, before a pressure

load is applied, require a less severe pressure stress for hypoxemic necrosis and ulceration to occur. It was further speculated that a contributing factor to the high incidence of pressure ulceration in certain areas of the body may be that the baseline skin circulation of these areas is decreased. The results of this study do not support this speculation. No significant differences were found between the PBCIs of the trochanter and groin. Therefore, decreased skin circulation when no pressure load has been applied does not appear to be a contributing factor to the high incidence of pressure ulceration over the trochanter. It is also possible that the thermal recovery method, and skin temperature measurement techniques do not adequately reflect the state of the cutaneous circulation.

2. Is the PBCI of the sacrum significantly greater than the PBCI of the abdomen?

No significant differences were found between the PBCIs of the sacrum and groin. As was the case with the trochanter and groin, researchers had assumed that the decreased skin temperature over the sacrum was due to decreased blood flow. The investigator speculated that decreased skin blood flow in skin which had not yet been subjected to a pressure load contributed to the high incidence of pressure ulceration over the

sacrum.

The results of this study indicate that there is no difference in rates of blood flow, as determined by the thermal recovery method, of skin over the sacrum and abdomen that has not been subjected to a pressure load. The higher pressure developed over the sacrum, leading to compression of blood vessels, is no doubt the major contributor to the development of pressure ulcers in this area.

No significant differences were found in the blood flow of skin not subjected to a pressure load when the trochanter was compared to the groin, or when the sacrum was compared to the abdomen. However, when a pressure load was applied, significant differences in the skin circulation of the susceptible and non-susceptible areas probably exist. Areas prone to pressure ulceration such as the trochanter and sacrum, are more often exposed to high pressure than other areas, such as the groin and abdomen. In addition, the anatomy of the sacral and hip areas (thin or non-existent layers of fat and muscle separating skin from bone) probably results in higher pressures being transmitted to the capillaries in these areas. Therefore, although there may not be a difference in skin circulation between areas prone to pressure ulceration

and adjacent, nonsusceptible areas before a pressure load is applied, under conditions of pressure stress, the skin circulation of the different areas probably differs markedly.

Additional Findings

Howell (1981) has reported the skin temperature values of areas prone to pressure ulceration, and of adjacent areas. Howell's findings are compared to those of the current study in Table 17. Since Howell reports only temperature ranges for the groin and abdomen, comparison of the two studies is difficult. Skin temperature values reported by Howell appear to be slightly higher at all sites than the temperature observed in the current study. This may be due to measurement error, or to differences in population or environment. The general trend in both studies is similar, with the groin being warmer than the trochanter, and the sacrum and abdomen having similar temperatures.

Howell explained the decreased skin temperature over the areas prone to pressure ulceration as being due to a less efficient circulation than in the adjacent areas. However, a number of factors other than skin blood flow may explain the difference in skin temperature. Skin temperature is determined by the rate of skin blood flow, the blood temperature, the thermal

Table 17

Baseline Skin Temperatures (° C): Howell (n=50)

and Present Study (n=30)

Site	Mean	Standard Deviation	Range
Trochanter			
Howell			
Right	33.05	1.28	28.2-35.2
Left	33.22	1.45	31.0-35.2
Present Study			
Right	32.32	1.22	29.4-34.0
Left	33.09	1.27	31.0-36.0
Groin			
Howell			
Right	-	-	33.0-36.8
Left	-	-	32.4-37.8
Present Study			
Right	33.90	1.00	31.8-35.4
Left	33.85	1.12	31.0-35.03
Sacrum			
Howell	34.96	0.61	33.4-36.0
Present Study	33.30	1.22	30.5-34.9
Abdomen			
Howell	-	-	33.0-36.8
Present Study	33.30	1.21	30.6-35.3

Note. Adapted from Howell, 1981.

conductivity of the tissue, the metabolic rate, and heat loss to the environment. When measuring skin temperature in one area of the body, factors affecting skin temperature other than blood flow can be controlled. However, when comparing different areas of the body, differences in thermal conductivity and metabolic rate may contribute greatly to differences in skin temperature. The results of the current study suggest that these skin temperature differences are not due to differences in skin blood flow, but to differences in metabolic rate of underlying tissue.

Limitations

Although the results of this study seem to indicate that there is no significant difference between the skin blood flow of areas prone to pressure ulceration, and adjacent, nonsusceptible areas, intervening variables may be responsible for these findings. Alternate explanations for the findings will be examined.

Three unanticipated patterns emerged which may cast doubt on the validity of the thermal recovery method as performed in this study.

1. The skin temperature readings frequently continued to decrease following removal of the cold glass container. It is unlikely that this is a

reflection of actual skin temperature. One possible explanation for this phemonenon is that the thermistors were unable to respond rapidly enough to the relatively large changes in skin temperature. Thermistors do not directly measure the temperature of the skin. The thermometer rather displays the temperature of the thermistor, which must stabilize with the temperature of the skin if there is to be an accurate reflection of skin temperature. In this study, thermistors were placed on the areas of study, and allowed to stabilize for five minutes before baseline skin temperatures were recorded. The thermistors were then moved to just outside the ten centimeter diameter circles centered over the site. The temperature of the thermistors thus remained fairly constant up to this point. After ice was applied to the skin for one minute, the thermistors were replaced in the center of the circles, and were subjected to a large drop in temperature. The rather large thermistors used in this study (2 mm, diameter) may have required several measurement periods to equalize with the skin temperature. Therefore, the lowest actual skin temperature may have been lower than the lowest recorded skin temperature. This inaccuracy would affect the calculation of the PBCI. Use of smaller thermistors with a more rapid

response time would increase the accuracy of similar studies in the future. Although the manufacturer states that the time constant of the thermistor is 1.1 seconds, and that 99% of a newly impressed temperature is read within five time constants, considerably longer than 5.5 seconds was required for temperatures to stabilize when the baseline skin temperatures were measured.

The continued decrease in skin temperature following the removal of the cold stimulus may also have been due to reflex vasoconstriction. Skin vessels respond to cold by constricting (Dodd, 1979). If this constriction is marked, skin blood flow would be interrupted following the removal of cold. With little or no heat flowing into the tissue, and heat loss to the environment continuing, the skin temperature could continue to decrease. The thermoregulatory vascular reflexes are unchanged by age in healthy persons. However, there is a group of elderly who show impaired regulation of skin blood flow and are at increased risk for developing hypothermia (Hanna & MacMillan, 1976). The reflex vasoconstriction in this group could be prolonged, and the skin temperature decrease following the removal of cold would be accentuated in this group.

Some antihypertensive medications effect peripheral vascular resistance and may have accentuated the skin temperature decrease. Clonidine, methyldopa, and hydralazine, taken by four subjects in this population, cause vasodilatation, and may have altered the skin's response to cold. The other antihypertensive medications taken by subjects in this study were beta blocking agents and diuretics, which probably do not have an effect on the peripheral circulation (Nickerson & Ruedy, 1975).

2. There was considerable variation between subjects in the initial decrease in skin temperature following the application of cold. The initial decrease in skin temperature ranged from 0.52^{O} C to 8.76^{O} C. This variation was most pronounced over the sacrum. The temperature decrease over the sacrum was significantly less than over the trochanter and abdomen, and approached significance when compared with the groin. Large variations in the initial decrease in skin temperature might result in variation in the PBCI, since large decreases in skin temperature would seem to result in slower thermal recovery, and small decreases to result in more rapid thermal recovery. However, when correlation coefficients were calculated between the initial decrease in skin

temperature and the PBCI, a significant correlation was noted only at the sacrum. If differences in the initial decrease in skin temperature affected the PBCI, this affect was apparent only at the sacrum. The sacrum differs from the other experimental areas in that there are no major blood vessels in the area, and there are no muscles or muscle insertions over the sacrum. These anatomical differences may have contributed to the differences in the initial decrease in temperature over the sacrum.

Intervening variables which could contribute to the differences in the initial decrease in skin temperature include: a) differences in the pressures used to apply the cold glass container to the skin, which could alter the thermal conductivity at the container-skin interface, and also cause changes in blood flow; b) differences in the application or adherence of thermistors to the skin, causing a difference in the thermal conductivity at the thermistor-skin interface; c) differences in the temperature of the container if the ice slurry did not cool the container to equal temperature; and d) physiologic and anatomic differences between subjects. Although the possible intervening variables were not measured, every effort was made to provide identical conditions

for each subject, and no differences in the pressure used to apply cold, the application or adherence of the thermistors, or the temperature of the container were noted by the investigator. Since the variation was greatest over the sacrum, it is possible that the anatomic differences of the sacrum caused most of this variation.

In order to determine the validity of the thermal recovery method as performed in this study, it is recommended that the study be repeated using another method of studying skin blood flow, such as laser Doppler velocimetry. Future studies using the thermal recovery method should include measurement of the temperature of the container, and if possible, measurement of the pressure used to apply the container to the skin. Strict attention should be paid to the adherence of thermistors to the skin.

3. The differences between the mean PBCIs of the trochanter and groin (16 seconds) and between the mean PBCIs of the sacrum and abdomen (19 seconds) were less than or equal to the accuracy of the measurement as performed in this study. From a cursory examination of the difference in mean PBCIs, it would appear that the thermal recovery method is not sufficiently sensitive to detect small differences in

blood flow.

However, when the absolute difference (numerical value without regard for positivity or negativity) between the PBCIs of the trochanter and groin was calculated for each subject, the PBCIs of the two sites were found to be identical in only two subjects, and differed by 20 seconds in only five subjects. Absolute differences in the PBCIs of the groin and trochanter within each subject ranged from 0 to 220 seconds, with a mean difference of 74.7 seconds.

Calculation of the absolute difference between the PBCIs of the sacrum and abdomen for each subject showed only two subjects with identical PBCIs at these sites. The PBCI differed by 20 seconds in only four subjects. The absolute difference in PBCI within each subject for these two sites ranged from 0 to 500 seconds, with a mean difference of 137 seconds.

Although the difference of mean PBCIs from site to site was small, large differences were noted on most individual subjects. The thermal recovery method appears to be adequately sensitive to detect site-to-site differences in skin blood flow.

No provision was made to control the number of calories consumed by the subjects prior to participation in the study, or to measure the insulation by

body fat, although these factors may influence body temperature. Since the thermal recovery method is more sensitive to variations in skin blood flow than simple measurement of skin temperature, these are probably not major limitations.

Small sample size may also have been a limitation in this study, contributing to type II error. Power is a statistical term used to indicate the probability of Type II error not occurring (Cohen, 1969). Assuming a difference in population means of 20 seconds, a population standard deviation of 100 seconds, and a one-tailed .05 decision rule, the power of thermal recovery method as performed in this study is about .30. In order to raise the power of the technique to .80, a sample size of 156 subjects would be required. Increasing the sample size may reveal a significant difference between the PBCIs at different sites. However, considering the large variation in PBCIs at individual sites, this seems unlikely.

Although every attempt was made to provide similar conditions of measurement for each subject, no measurements were made of the pressure applied to the skin by the cold glass container, or of the temperature of the container. Small differences in these values may have altered the PBCIs of subjects. The thermistors

were applied in an identical manner to each subject, but small changes in position and muscle movement may have caused displacement of the thermistor probes, resulting in erroneous values. Another source of erroneous temperature values may have been the relatively slow response time of the thermistors.

No attempt was made to control ambient temperature or humidity. The room temperature varied only 2^{O} C during the course of the study, and no change in room temperature was noted during the course of measurement of any one subject, so this was probably not a major limitation. Measurements were made between one pm and four-thirty pm. Diurnal variations in body metabolism may have accounted for variations in skin temperature. No attempt was made to control for antihypertensive medications.

Implications for Nursing Practice

Although the validity of the thermal recovery method, as performed in this study, may be questioned, the data indicates that there is no significant difference between the skin blood flow of areas prone to pressure ulceration and adjacent, nonsusceptible areas under baseline conditions.

A long-standing nursing belief is that massage

of bony prominences is useful in the prevention of pressure ulcers. As usually practiced, the bony prominences are massaged after the patient has been repositioned so that pressure is no longer applied to the area, especially if the skin is reddened. The rationale for this procedure is: a) pressure ulcers are due to interruption of blood flow caused by pressure; b) reddening of the skin indicates that excessive pressure has been applied; and c) massage of the skin increases local circulation and has a protective effect.

Reddening of the skin indicates that vasodilatation and increased blood flow have occurred. This reactive hyperemia also occurs in other tissues. Even though no reddening of the skin may be noted, the circulation of underlying tissue may be increased following the release of pressure. It is doubtful that measures to increase blood flow would have a protective effect in this case. Massage of bony prominences to increase skin blood flow before the application of a pressure load may be more beneficial in preventing pressure ulcers.

Research has shown conclusively that prolonged, excessive pressure is the primary contributor to the development of pressure ulcers, but has not shown

that variations in baseline skin blood flow contribute to their development. Therefore, nursing intervention should continue to concentrate on decreasing the amount and duration of pressure applied to the surface of the body. Measures to increase the local circulation are of dubious value.

CHAPTER V

SUMMARY AND RECOMMENDATIONS

Summary

Pressure ulcers are areas of localized tissue necrosis caused by prolonged, high, externally-applied pressure which limits local blood flow, resulting in anoxia and necrosis.

Pressure ulcers occur most commonly in areas where high pressure develops, such as over the trochanters and sacrum. The anatomy of these areas differs from other nonsusceptible areas in that the bony prominences are covered by relatively thin layers of muscle or fat. This lack of "padding" causes higher pressures to develop in these areas.

The skin vasculature is supplied by perforating arteries, which penetrate muscle and give off branches. Little information is available regarding the local skin circulation in areas prone to pressure ulceration. Decreased baseline skin blood flow could contribute to the susceptibility of these areas to pressure ulceration.

Little research has been conducted to determine the baseline skin blood flow in areas prone to pressure ulceration. A number of studies have examined the skin temperature of these areas. The skin temperature of unstressed areas prone to pressure ulceration was generally found to be cooler than that of other areas. Some investigators have concluded that the coolness is due to decreased skin circulation. However, skin temperature is determined by heat production of underlying tissues, thermal conductivity of the tissue, and heat loss to the environment, in addition to skin blood flow. Therefore, a more sensitive method of studying skin blood flow is required.

Two studies utilizing clearance of radioactive substances to study the skin circulation of these areas were inconclusive and contradictory.

The thermal recovery method is more sensitive to variations in skin blood flow than simple measurement of skin temperature. The peripheral blood circulatory index (PBCI), or the time required for the skin temperature to return half-way to normal following the application of cold, is an index of skin circulation. It has been shown to correlate well with clearance of radioactive xenon as a measure of skin circulation.

The purpose of this study was to determine whether

there were differences in the baseline skin circulation, as measured by the thermal recovery method, between areas prone to pressure ulceration, and adjacent non-susceptible areas.

The specific research questions asked were:

1. Is the PBCI over the trochanter greater than the PBCI over the groin?

2. Is the PBCI over the sacrum greater than the PBCI over the abdomen?

No significant differences were found between the PBCIs of any of these areas. Although a number of limitations of the study were present, the data points to a lack of difference between the skin circulation of susceptible and nonsusceptible areas.

Nursing implications include concentrating nursing intervention on reducing magnitude and duration of pressure applied to the body, rather than on attempting to increase the local skin circulation, which is of unproven value.

Recommendations for Further Research

Research recommendations for further research utilizing the thermal recovery method include:

1. Using smaller thermistors with a more rapid response time.

2. Quantifying potential intervening variables

such as the pressure used to apply the container of the skin and the temperature of the container.

3. Emphasizing secure attachment and adherence of thermistors to the skin.

Research recommendations for further research to determine the role of local skin blood flow to the development of pressure ulcers include:

1. Repeating the study utilizing another technique of measuring skin blood flow, such as laser doppler velocimetry, to overcome the limitations of the thermal recovery method and validate the results of the study.

2. Determining quantitative changes in skin blood flow following the use of various modalities to increase skin blood flow such as massage, heat and application of topical agents. Measuring skin blood flow with the thermal recovery method or another technique before and after use of the modality.

3. Determining if those modalities which increase skin blood flow continue to do so after application of a pressure load. Measuring skin blood flow before and after use of the modality and after the application of the pressure load. Comparing to a control group where a pressure load is applied, but no modality to increase skin blood flow is used.

4. Performing animal studies to determine if increasing skin blood flow is useful in the prevention of pressure ulcers. Increasing blood flow using appropriate modality and determining the magnitude and duration of pressure required to produce an ulcer, and then comparing this to the duration and magnitude of pressure required to produce an ulcer in a control group where no modality was used.

APPENDIX A

INFORMED CONSENT FORM

I _____ agree to serve as a subject in the investigation "Local Skin Circulation as Measured by the Thermal Recovery Method in Areas Prone to Pressure Ulceration and Adjacent Areas," conducted by Vern Hedegaard, R.N., Master of Science Candidate, University of Utah College of Nursing. The purpose of this study is to obtain information about the contribution of local blood flow to the development of pressure ulcers or bedsores.

The procedures to which I will be subjected include:

1. Application of an ice filled glass container to the skin over the hips, tailbone, groin, and abdomen for one minute.

2. Measurement of skin temperature using an electrical thermometer with a stainless steel button taped to the skin at the above sites and at the opposite groin and right arm.

The time required for these measurements is approximately one hour. The methods of measurement have been used before and do not involve introducing anything into the body. There may be some discomfort associated with application of the cold glass container to the skin, but no substantial risks are anticipated. I understand that there may be no benefits to me personally, but that this information may lead to better methods of treating and preventing bedsores in the future.

I understand that the data collected will be presented in grouped form to assure confidentiality.

I am not receiving any compensation for my participation in this study. I understand that I can withdraw from this study at any time without prejudice or penalty.

I have read the above and have had the opportunity to ask questions and receive answers.

Signature _____

Date _____

APPENDIX B

THERMOMETER AND THERMISTOR SPECIFICATIONS

Thermometer: Digitec

Model: 5810

Two Ranges: -30.00 C to +50.00 C or 0.00 C to 100.00 C

Resolution: 0.01 C

Accuracy at 23 C + 3 C 85% r.h.: ± 0.42 C for the -30.00 C to +50.00 C range; ± 0.45 C for the 0.00 C to 100.00 C range

Probe Inputs: Three sets on the front panel

Polarity Indication: ± LED indicator, displayed automatically

Response Time: 500 mSec

Sampling Rate: 4/sec nominal

Temperature Coefficient: 0.01 C/C

Operating Ambient: 0 C to 50 C

Display: LED 0.4" high

Power Requirements: 115/230 v AC, 50/400 Hz, 2.5 vA

Battery Capacity: 7 hours typical continuous operation; 16 hours recharge from full discharge

Weight: Net 2 pounds

Thermistor Probes: Yellow Springs Instruments

Model Number: 709A

Accuracy: ± 0.15 C from -30 C to 100 C

Time Constant: 1.1 seconds

Description: Attachable surface temperature. Stainless steel cup, epoxy backed. Easy to tape to flat surfaces. Ten foot lead attached to stainless steel cup.

Time Constant: The time required for the probe to read 63% of a newly impressed temperature. Approximately five time constants are required for a probe to read 99% of the total change.

APPENDIX C

DATA COLLECTION SHEETS

Date _____

Time _____

Name _____

Subject Number _____

Height _____

Weight _____

Sex _____

Blood Pressure _____

Medical Conditions: Diabetes _____

Hypertension _____

Peripheral Vascular Disease _____

Surgical or Traumatic Scars _____

Prior History of Pressure Ulcers _____

Medications: Antihypertensive Medications _____

Anticoagulants _____

Other _____

Trochanter to be Studied _____

Name _____ Date _____ Time _____

Room Temp Pre _____ Room Temp Post _____

Trochanter

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Groin

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Groin (Contralateral)

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Right Brachial

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Name _____

Sacrum

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Abdomen

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

Right Brachial

Pre	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

APPENDIX D

PROTOCOL

The Protocol

1. Plug thermometer into outlet, plug thermistors into thermometer.

2. Fill glass containers with ice-water slurry.

3. Approach subject in waiting area, introduce self, give brief explanation of study, and ask subject to participate.

4. Obtain subject's signature on informed consent form.

5. Weigh subject - clothed, shoes off.

6. Measure subject's height.

7. Escort subject to exam room. Have subject remove outer clothing and put on patient gown. If subject wears LDS garments, have subject remove.

8. Record room temperature.

9. Randomly select trochanter to be studied. Have subject lie on contralateral side.

10. Mark ten centimeter diameter circles over trochanter and ipsilateral groin.

11. Apply thermistor #1 to center of circle over trochanter with paper tape.

12. Apply thermistor #2 to center of circle over ipsilateral groin with paper tape.

13. Apply thermistor #3 to contralateral groin.

14. Apply thermistor #4 to right brachial region.

15. Insulate thermistors with cotton balls held in place with paper tape.

16. Allow five minutes to elapse. During this time perform steps 17 and 18.

17. Measure subject's blood pressure.

18. Interview subject to obtain age, medical conditions, and medications taken.

19. Record the initial skin temperatures from thermistors 1, 2, 3, and 4.

20. Move thermistor #1 and attach to skin just outside circle over trochanter.

21. Move thermistor #2 and attach to skin just outside circle over ipsilateral groin.

22. Gently apply ice water slurry filled containers to skin inside circles over trochanter and ipsilateral groin for one minute. Apply only enough pressure to hold the containers in place.

23. Reattach thermistors 1 and 2 to skin in centers of circles over trochanter and ipsilateral groin.

24. Record skin temperature from thermistors 1, 2, 3, and 4.

25. Continue recording skin temperatures from all four thermistors at 20 second intervals for 19 more measurement periods.

26. If, after 20 measurement periods, the skin temperature of the trochanter and groin have not returned half-way to the original skin temperature, continue recording skin temperature from all four thermistors at 20 second intervals until the temperature over the groin and trochanter has reached this point.

27. Remove thermistors 1, 2, and 3 from skin.

28. Remove markings from skin with nail polish remover.

29. Mark ten centimeter circles over sacrum and abdomen.

30. Apply thermistor #1 to center of circle over sacrum. Attach with paper tape.

31. Apply thermistor #2 to center of circle over abdomen. Attach with paper tape.

32. Insulate thermistors with cotton balls held in place with paper tape.

33. Leave thermistor #4 in place over right brachial region.

34. Allow five minutes for stabilization of thermistors.

35. Move thermistors 1 and 2 and attach to skin just outside circles over sacrum and abdomen.

36. Apply cold glass containers to skin inside circles over sacrum and abdomen for one minute.

37. Reattach thermistors 1 and 2 to skin in center of circles over sacrum and abdomen.

38. Record skin temperature from thermistors 1, 2, and 4.

39. Record skin temperature from all three thermistors at 20 second intervals. Continue recording skin temperature for 19 more measurement periods or until the skin temperature over the sacrum and abdomen has returned half-way to the initial temperature, whichever comes later.

40. Remove thermistors from skin.

41. Remove markings from skin with nail polish remover.

42. Thank subject.

APPENDIX E

MEAN TEMPERATURE VALUES

Trochanter

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	32.702	1.28	29.35-36.00
2	30	27.71	1.40	23.96-29.58
3	30	27.44	1.33	24.00-29.83
4	30	27.60	1.29	24.28-29.83
5	30	27.89	1.28	26.63-30.21
6	30	28.19	1.29	24.93-30.8
7	30	28.48	1.29	25.27-31.30
8	30	28.75	1.29	25.54-31.74
9	30	29.21	1.31	26.05-32.60
10	30	29.21	1.31	26.05-32.60
11	30	29.39	1.32	26.32-32.95
12	30	29.57	1.32	26.52-33.25
13	30	29.75	1.33	26.78-33.53
14	30	29.90	1.35	26.94-33.82
15	30	30.06	1.36	27.12-34.05
16	30	30.20	1.36	27.32-34.27
17	30	30.33	1.37	27.49-34.46
18	30	30.45	1.38	27.62-34.63
19	30	30.56	1.38	27.71-34.79
20	29	30.62	1.38	27.82-34.91
21	28	30.74	1.40	27.93-35.03
22	16	30.31	1.23	28.05-32.57
23	12	30.39	1.35	28.18-32.64
24	11	30.27	1.20	28.29-32.30
25	10	30.17	1.06	28.33-32.10
26	8	30.08	1.15	28.35-32.17
27	7	29.85	0.86	28.39-31.10

Trochanter (Continued)

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
28	7	29.92	0.88	28.42-31.22
29	3	29.39	0.83	28.45-30.03
30	2	29.89	0.22	29.73-30.05
31	1	29.79		

Groin

Observation Number	Number of Observations	Mean Temperature (° C)	Standard Deviation	Range
1	30	33.88	0.96	31.20-35.37
2	30	29.25	1.60	31.14-35.45
3	30	29.19	1.55	31.10-35.49
4	30	29.42	1.52	30.84-35.51
5	30	29.66	1.47	30.56-35.53
6	30	29.93	1.42	30.20-35.56
7	30	30.20	1.38	29.38-35.58
8	30	30.43	1.35	30.67-35.61
9	30	30.65	1.34	31.01-35.62
10	30	30.85	1.31	31.17-35.64
11	30	31.01	1.30	31.25-35.66
12	30	31.16	1.28	31.30-35.69
13	30	31.30	1.27	31.33-35.70
14	30	31.43	1.27	31.34-35.73
15	30	31.55	1.26	31.37-35.75
16	30	31.68	1.27	31.39-35.77
17	30	31.78	1.28	31.40-35.80
18	30	31.88	1.28	31.42-35.83
19	30	31.98	1.29	31.43-35.85
20	29	31.99	1.25	31.43-35.85
21	28	32.07	1.28	31.40-35.89
22	16	31.63	1.22	32.73-35.78
23	12	31.88	1.29	32.76-35.79
24	10	32.14	1.09	32.76-35.80
25	9	32.20	1.16	32.77-35.81
26	8	32.12	1.18	32.78-35.81
27	6	32.11	1.28	31.50-35.20

Groin (Continued)

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
28	7	32.07	1.18	30.03-33.64
29	3	31.27	1.09	30.05-32.14
30	1	32.18		
31	1	32.24		

Contralateral Groin

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	33.87	1.15	31.20-35.37
2	30	34.04	1.14	31.14-35.45
3	30	34.04	1.14	31.10-35.49
4	30	34.04	1.17	30.84-35.51
5	30	34.05	1.21	30.56-35.53
6	30	34.05	1.25	30.20-35.56
7	30	34.03	1.35	29.38-35.58
8	30	34.08	1.22	30.67-35.61
9	30	34.10	1.19	31.01-35.62
10	30	34.11	1.18	31.17-35.64
11	30	34.12	1.18	31.25-35.61
12	30	34.13	1.18	31.30-35.69
13	30	34.14	1.18	31.33-35.70
14	30	34.15	1.18	31.34-35.73
15	30	34.15	1.18	31.37-35.74
16	30	34.16	1.18	31.39-35.77
17	30	34.17	1.19	31.40-35.80
18	30	34.17	1.18	31.42-35.83
19	30	34.17	1.19	31.43-35.85
20	29	34.13	1.19	31.43-35.85
21	28	34.13	1.22	31.40-35.89
22	16	34.14	0.94	32.73-35.78
23	12	34.18	0.97	32.76-35.79
24	11	34.26	0.97	32.76-35.80
25	8	34.20	1.02	32.77-35.81
26	8	34.05	1.05	32.78-35.81
27	7	33.51	1.19	31.50-35.20

Contralateral Groin (Continued)

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
28	7	33.78	0.82	32.80-35.20
29	3	33.56	0.92	32.80-34.58
30	2	33.94	0.92	33.29-34.59
31	1	33.94		

Right Brachial Phase I

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	33.19	1.16	31.20-35.41
2	30	33.50	1.14	31.30-35.77
3	30	33.50	1.15	31.24-35.80
4	30	33.55	1.13	31.66-35.82
5	30	33.57	1.13	31.65-35.83
6	30	33.59	1.14	31.58-35.84
7	30	33.63	1.15	31.52-35.85
8	30	33.63	1.14	31.59-35.87
9	30	33.66	1.14	31.53-35.89
10	30	33.71	1.17	31.44-35.91
11	30	33.72	1.18	31.45-35.93
12	30	33.72	1.17	31.42-35.97
13	30	33.74	1.17	31.38-35.98
14	30	33.76	1.17	31.36-35.98
15	30	33.78	1.17	31.34-35.98
16	30	33.80	1.18	31.29-35.97
17	30	33.81	1.19	31.10-35.99
18	30	33.83	1.20	31.06-36.01
19	30	33.85	1.20	31.01-36.01
20	29	33.88	1.23	30.98-36.01
21	28	33.90	1.25	30.97-36.01
22	17	33.43	1.14	30.95-35.14
23	12	33.60	1.18	30.95-35.14
24	11	33.47	1.23	30.94-35.12
25	9	33.46	1.37	30.94-35.10
26	8	33.75	1.08	32.15-35.09
27	7	33.66	1.12	32.06-35.12

Right Brachial Phase I

(Continued)

Observation Number	Number of Observation	Mean Temperature (° C)	Standard Deviation	Range
28	7	33.64	1.10	32.07-35.16
29	2	33.47	1.42	32.47-34.48
30	1	34.53		
31	1	34.58		

Sacrum

Observation Number	Number of Observation	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	33.32	1.22	30.54-34.92
2	30	29.43	1.79	25.96-32.34
3	30	29.36	1.87	25.30-32.33
4	30	29.42	1.82	25.36-32.33
5	30	29.59	1.82	25.42-32.62
6	30	28.82	1.75	25.47-32.97
7	30	29.92	1.77	25.53-33.25
8	30	30.09	1.74	25.62-33.44
9	30	30.24	1.73	25.73-33.66
10	30	30.41	1.71	25.83-33.85
11	30	30.525	1.71	25.93-34.01
12	30	30.67	1.69	26.03-34.17
13	30	30.80	1.68	26.15-34.28
14	30	30.94	1.67	26.24-34.42
15	30	31.14	1.81	26.30-34.98
16	30	31.15	1.66	26.38-34.64
17	30	31.29	1.69	26.43-34.75
18	30	31.36	1.67	26.52-34.83
19	30	31.45	1.67	26.60-34.90
20	29	31.53	1.67	26.66-34.97
21	29	31.64	1.67	26.75-35.02
22	22	31.44	1.62	26.83-34.06
23	18	31.39	1.68	26.90-34.20
24	15	31.14	1.60	26.96-33.67
25	13	31.25	1.71	27.03-33.71
26	13	31.33	1.70	27.08-33.75
27	12	31.31	1.72	27.16-33.79

Sacrum (Continued)

Observation Number	Number of Observation	Mean Temperature (° C)	Standard Deviation	Range
28	11	31.17	1.59	27.25-33.51
29	7	30.71	1.54	27.35-31.63
30	7	30.78	1.54	27.42-31.70
31	6	30.74	1.67	27.46-31.77
32	4	30.33	1.92	27.54-31.65
33	3	29.95	2.10	27.59-31.62
34	3	29.30	2.08	27.66-31.64
35	3	30.06	2.02	27.79-31.66
36	3	30.09	2.01	27.82-31.67
37	3	30.12	2.01	27.85-31.68
38	3	30.14	2.00	27.88-31.70
39	2	29.82	2.67	27.93-31.71
40	2	29.84	2.66	27.96-31.72
41	2	29.86	2.64	28.00-31.73
42	1	28.05		
43	1	28.09		

Abdomen

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	33.34	1.21	30.63-35.27
2	30	28.87	1.94	24.30-31.88
3	30	28.78	1.87	24.37-32.01
4	30	28.93	1.78	24.66-32.14
5	30	29.14	1.71	25.24-32.20
6	30	29.35	1.66	25.77-32.25
7	30	29.58	1.60	26.41-32.34
8	30	29.77	1.56	26.72-32.40
9	30	29.93	1.54	27.08-32.49
10	30	30.14	1.52	27.46-32.59
11	30	30.28	1.48	27.74-32.69
12	30	30.43	1.46	27.93-32.74
13	30	30.57	1.45	28.09-32.84
14	30	30.70	1.44	28.31-33.26
15	30	30.80	1.43	28.47-33.40
16	30	30.93	1.43	28.55-33.78
17	29	31.14	1.39	29.13-34.01
18	29	31.23	1.39	29.22-34.12
19	29	31.32	1.39	29.28-34.25
20	29	31.39	1.40	29.31-34.36
21	29	31.50	1.38	29.36-34.42
22	22	31.215	1.17	29.38-33.38
23	18	31.27	1.22	29.42-33.46
24	17	31.28	1.21	29.48-33.53
25	13	31.31	1.29	29.51-33.60
26	13	31.27	1.27	29.61-33.68
27	12	31.25	1.19	29.64-33.73

Abdomen (Continued)

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
28	11	31.10	0.92	29.69-32.50
29	6	31.07	1.00	30.00-32.52
30	5	31.31	0.98	30.04-32.54
31	5	31.17	0.94	30.05-32.50
32	3	30.73	0.72	29.99-31.25
33	3	30.67	0.69	30.02-31.32
34	2	30.46	0.78	30.08-30.84
35	2	30.84	0.64	30.15-31.53
36	2	30.87	0.62	30.21-31.53
37	2	30.92	0.63	30.26-31.68
38	2	30.98	0.61	30.35-31.61
39	2	31.01	0.59	30.40-31.71
40	2	31.06	0.58	30.49-31.63
41	2	31.11	0.58	30.55-31.67
42	2	31.20	0.48	30.64-31.76
43	1			

Right Brachial Phase Two

Observation Number	Number of Observations	Mean Temperature (^{O} C)	Standard Deviation	Range
1	30	33.87	1.12	31.09-35.29
2	30	33.73	1.24	31.22-35.04
3	30	33.73	1.25	31.14-35.07
4	30	33.74	1.25	31.13-35.01
5	30	33.77	1.27	31.10-35.07
6	30	33.78	1.27	31.10-35.07
7	30	33.79	1.27	31.09-35.06
8	30	33.80	1.28	31.07-35.05
9	30	33.76	1.26	31.03-35.08
10	30	33.70	1.27	30.97-35.10
11	30	33.67	1.28	30.94-35.13
12	30	33.69	1.29	30.92-35.15
13	30	33.74	1.29	30.94-35.18
14	30	33.78	1.24	31.22-35.19
15	30	33.80	1.18	31.27-35.21
16	30	33.81	1.16	31.25-35.23
17	29	33.77	1.14	31.25-31.15
18	29	33.77	1.13	31.26-35.17
19	29	33.78	1.11	31.25-35.19
20	29	33.79	1.11	31.25-35.21
21	29	33.81	1.10	31.23-35.22
22	21	33.56	1.12	31.23-35.23
23	18	33.63	0.98	32.21-35.23
24	17	33.65	1.01	32.23-35.23
25	13	33.92	0.96	32.29-35.24
26	13	33.92	0.95	32.35-35.24
27	12	33.91	0.99	32.31-35.23

Right Brachial Phase Two

(Continued)

Observation Number	Number of Observations	Mean Temperature (C C)	Standard Deviation	Range
28	11	34.06	0.89	32.39-32.24
29	7	34.20	0.85	33.32-35.25
30	5	34.16	1.00	33.35-35.25
31	4	34.33	1.07	33.37-35.26
32	2	34.36	1.28	33.46-35.26
33	2	34.36	1.27	33.47-35.26
34	2	34.37	1.27	33.47-35.27
35	2	34.37	1.27	33.48-35.27
36	2	34.37	1.24	33.49-35.25
37	2	34.37	1.24	33.50-35.25
38	2	34.34	1.25	33.46-35.23
39	2	34.33	1.24	33.45-35.21
40	2	34.32	1.23	33.45-35.19
41	2	34.31	1.21	33.46-35.17
42	2	34.32	1.20	33.47-35.17
43	1	33.46		

REFERENCES

Argis, J. & Spira, M. (1979). Pressure ulcers: Prevention and treatment. Clinical Symposia, 31, 1-32.

Baptista, A. (1970). A simple thermal method for the study of the peripheral blood circulation and applications, Microvascular Research, 2, 123-138.

Barbanel, J., Jordan, M. & Nichol, S. (1977). Incidence of pressure- cores in the greater Glasgow health board area. The Lancet, 2(806), 548-550.

Barton, A. (1973). Pressure scores viewed by electron microscope and thermography. Geriatrics, 28, 143-147.

Beland, I. & Passos, J. (1975). Clinical nursing: Pathophysiological and psychosocial approaches (3rd Ed.). New York: MacMillan.

Bennett, L., Kavner, D., Lee, B. & Trainor, F. (1979). Shear vs. pressure as causative factors in skin blood flow occlusion. Archives of Physical Medicine and Rehabilitation, 60, 309-314.

Beyers, M. & Dudas, S. (1977). The clinical practice of medical-surgical nursing. Boston: Little, Brown and Company.

Brooks, B. & Duncan, G. (1940). Effects of pressure on tissues. Archives of Surgery, 40, 696-711.

Brunner, L. & Suddarth, D. (1974). (Eds.). The Lippincott manual of nursing practice. Philadelphia: Lippincott.

Brunner, L., Emerson, C., Ferguson, L. & Suddarth, D. (1974). (Eds.). Textbook of medical-surgical nursing (2nd Ed.). Philadelphia: Lippincott.

Cattell, M. (1936). The physiological effects of pressure, Cambridge Philosophic Society Reviews, 11, 441-483.

Cohen, J. (1969). Statistical power analysis for the behavioral sciences. New York: Academic Press.

Daniel, R., Priest, D. & Wheatley, D. (1981). Etiologic factors in pressure sores: An experimental model. Archives of Physical Medicine and Rehabilitation, 54, 51-56.

Dinsdale, S. (1973). Decubitus ulcers in swine: Light and electron microscopy study of pathogenesis. Archives of Physical Medicine and Rehabilitation, 54, 51-56.

Dinsdale, S. (1974). Decubitus ulcers: Role of pressure and friction in causation. Archives of Physical Medicine and Rehabilitation, 55, 147-152.

Dodd, M. (1979). Caring for persons requiring therapeutic application of heat and cold. In Sorenson, K. & Luckman, J. (Eds.), Basic nursing: A psychophysiologic approach. Philadelphia: Saunders.

Exxon-Smith, A. (19). The prevention of pressure sores: Significance of spontaneous bodily movements, The Lancet, 2(7213), 1124-1126.

Galpin, J., Chow, A., Bayer & Guze, L. (1976). Sepsis associated with decubitus ulcers. The American Journal of Medicine, 61, 346-350.

Goldblatt, H. (1926). Reactive hyperemia. Heart, 12, 281-294.

Goller, H., Lewis, D. & McLaughlin, R. (1971). Thermographic studies of human skin subjected to localized pressure. American Journal of Roentgenology, Radium, Thermographic, and Nuclear Medicine, 113, 749-754.

Goller, H., Lewis, D., McLaughlin, R. & Verhonick, P. (1976). The effect of external pressure on skin temperature distribution by thermograph. Medical Research Engineering, 12, 6-8.

Griffith, R. (1963). Advances in the treatment of decubitus ulcers. Surgical Clinics of North America, 43, 245-260.

Helleway, G. (1980). Blood flow responses to injection trauma measured by laser doppler velocimetul. Journal of Investigative Dermatology, 74, 1-4.

Holloway, G., Dalt, C., Kennedy, D. & Chimoskey, J. (1976). Effects of external pressure loading on human skin blood flow measured by ^{133}Xe clearance. Journal of Applied Physiology, 40, 597-600.

Holloway, G. & Watkins, D. (1977). Laser-doppler measurement of cutaneous blood flow. Journal of Investigative Dermatology, 69, 306-307.

Howell, T. (1981). Skin temperature of bedsore areas in the aged. Experimental Gerontology, 16, 137-140.

Hussain, T. (1953). An experimental study of some pressure effects on tissue, with reference to the bed-sore problem. Journal of Pathology and Bacteriology, 66, 347-359.

Isiandinso, O. (1979). Decubitus ulcers in geriatric patients: Present status. New York State Journal of Medicine, 79, 2027-2029.

Kirkendall, W., Burton, A., Epstein, F. & Freis, E. (1967). Committee of the American Heart Association: Recommendations for human blood pressure measurement by sphygmomanometers. Circulation, 36, 980-988.

Kosiak, M. (1959). Etiology and pathology of ischemic ulcers. Archives of Physical Medicine and Rehabilitation, 40, 62-69.

Kosiak, M. (1961). Etiology of decubitus ulcers. Archives of Physical Medicine and Rehabilitation, 42, 19-29.

Kosiak, M., Kubicek, W., Olsen, M., Danz, J. & Kottke, F. (1958). Evaluation of pressure as a factor in the production of ischial ulcers. Archives of Physical medicine and Rehabilitation, 39, 623-629.

Krog, J. (1962). Electrical measurement of body temperature. Acta Anesthesia Scandinavia (Supplement), 11, 99-113.

Landis, E. (1930). Microinjection studies of capillary blood pressure in human skin. Scandinavian Journal of Plastic and Reconstructive Surgery, 15, 209-228.

Landis, B., Holstein, P. & Lassen, N. (1979). On the pathogenesis of bedsores. Scandinavian Journal of Plastic and Reconstructive Surgery, 13, 347-350.

Larsen, B., Holstein, P. & Lassen, N. (1979). On the pathogenesis of bedsores. Scandinavian Journal

of Plastic and Reconstructive Surgery, 13, 347-350.

Lewis, T. & Grant, R. (1925). Observations upon reactive hyperemia. Heart, 12, 73-120.

Lewis, T. & Landis, E. (1929). Some physiological effects of sympathetic ganglionectomy in the human being and its effect in a case of Reynaud's malady. Hearts, 15, 151-176.

Linden, O. (1961). Etiology of decubitus ulcers: An experimental study. Archives of Physical Medicine and Rehabilitation, 42, 774-783.

Mackay, R. (1970). Biomedical telemetry (2nd ed.), New York: John Wiley and Sons.

Mahanty, S. & Roemer, R. (1979). Thermal response of skin to application of localized pressure. Archives of Physical Medicine and Rehabilitation, 60, 584-590.

Mahanty, S. & Roemer, R. (1980). Therman and circulatory response of tissue to localized pressure application: A mathematical model. Archives of Physican Medicine and Rehabilitation, 61, 335-340.

Martin, D. (1971). The medical patient. In Moidel, H., Sorenson, G., Giblin, E. & Kaufman, M. (Eds.), Nursing care of patients with medical-surgical disorders. New York: McGraw-Hill.

McClemont, E., Shand, I. & Ramsay, B. (1977). Pressure sores: A new method of treatment. The British Journal of Clinical Practice, 33, 21-25.

McClennan, C., McClennan, M. & Landis, E. (1942). The effect of external pressure on the forearm and its relation to capillary blood pressure and venous pressure. Journal of Clinical Investigation, 12, 319-338.

Montagna, W. & Prakkal, P. (1974). The structure and function of the skin, New York: Academic Press.

Newman, P. & Davis, N. (1981). Thermography as a predictor of sacral pressure sores. Age and Aging, 10, 14-18.

Nickerson, M. & Ruedy, J. (1975). Antihypertensive

agents and the drug therapy of hypertension. In Goodman, L. & Gilman, A. (Eds.), The pharmacological basis of therapeutics. New York: MacMillan.

Nola, G. & Vistnew, L. (1980). Differential response of skin and muscle in the experimental production of sores. Plastic and Reconstructive Surgery, 66, 728-733.

Palmer, J. (1979). Shear vs. pressure measurement. Archives of Physical Medicine and Rehabilitation, 60, 614-615.

Patterson, R. & Fisher, S. (1980). Pressure and temperature patterns under the ischial tuberosities. Bulletin of Prosthetics Research, 17, 5-11.

Peterson, N. & Bittman, S. (1971). The epidemiology pressure sores. Scandinavian Journal of Plastic and Reconstructive Surgery, 5, 62-64.

Polit, D. & Hungler, B. (1978). Nursing research: Principles and methods. Philadelphia: Lippincott.

Reuler, J. & Cooney, T. (1981). The pressure sores: Pathophysiology and principles of management. Annals of Internal Medicine, 94, 661-666.

Richardson, R. & Meyer, P. (1981). Prevalence and incidence of pressure sores in acute spinal cord injuries. Paraplegia, 19, 235-247.

Roberts, B. & Goldstone, L. (1979). A survey of pressure sores in the over sixties on two orthopedic wards. International Journal of Nursing Studies, 16, 355-364.

Ryan, T. (1973). Structure, pattern, and shape of the blood vessels of the skin. Measurement of blood flow and other properties of the skin. In Jarrett, A. (Ed.), The physiology and pathophysiology of the skin (Vol. 2). New York: Academic Press.

Ryan, T. (1976). The blood vessels of the skin. The Journal of Investigative Dermatology, 67, 110-118.

Schell, V. & Wolcott, L. (1966). The etiology,

prevention, and treatment of decubitus ulcers. Missouri Medicine, 63, 109-119.

Stoll, A. & Hardy, J. (1950). Study of thermocouples as skin thermometers. Journal of Applied Physiology, 11, 531-537.

Tavares, C., Ferretra, J. & Fernandes, F. (1975). Cutaneous blood flow: A comparative study between the thermal recovery method and the radioxenon clearance method. Bibliographica Anatomica, 23, 137-155.

Trandel, A., Lewis, D. & Verhonick, P. (1975). Thermographical investigation of decubitus ulcers. Bulletin of Prosthetics Research, 23, 137-155.

Verhonick, P., Lewis, D. & Goller, H. (1972). Thermography in the study of decubitus ulcers: Preliminary report. Nursing Research, 21, 233-237.

Williams, A. (1972). A study of factors contributing to skin breakdown. Nursing Research, 21, 238-243.